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BLOOD LACTIC ACID CONCENTRATIONS WITH VARYING EXERCISE INTENSITIES

by

© LYNTON JOHN DAY

A THESIS

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies for acceptance, a thesis entitled "Blood Lactic Acid Concentrations with Varying Exercise Intensities" submitted by Lynton John Day in partial fulfilment of the requirements for the degree of Master of Arts.

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ABSTRACT

The hypothesis tested in this study was that elevated blood lactic acid levels could be lowered in exercise, provided that the level of exercise remained aerobic.

Ten subjects performed three treatment conditions. In treatment I they rode on a bicycle ergometer for 90 seconds at 90 percent MVO_2, then for 15 minutes at 30 percent MVO_2 finishing at 90 percent MVO_2 for 90 seconds. Treatment II consisted of 55 percent MVO_2 for 90 seconds, 30 percent MVO_2 for 15 minutes and 90 percent MVO_2 for 90 seconds. Treatment III was 90 percent MVO_2 for 90 seconds. Blood samples were taken from the brachial artery at 5, 10 and 14 minutes of exercise and at 2, 5, and 8 minutes of recovery and expired gas was collected for 20 minutes after exercise.

Comparisons of the lactate levels attained throughout and at the conclusion of each treatment were made graphically. Definite trends were apparent which supported the following conclusions: (1) Blood lactate levels do not constantly increase in exercise at a rate proportional to the metabolic rate. They may be reduced in aerobic exercise to approximately resting levels; (2) lactate metabolism is faster in exercise than at rest; (3) No relationship appeared to exist between lactate levels and oxygen debt; and (4) increase in lactate provides the same relative information to the investigator as the excess lactate measure.

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CHAPTER I

INTRODUCTION

Lactic acid can be a limiting factor in exercise if its concentration in the blood reaches a critical level. The law of Mass Action states that as the end products of a chemical reaction build up in the reacting medium, the rate of the reaction approaches zero. In the case of lactic acid, the blood pH decreases and unconsciousness can result.

It had early been realized (17) that lactic acid produced during muscular contraction was formed from the glycogen stored in the muscle and that these reactions formed the major energy source during anaerobic contraction. Following this, A.V. Hill (23) in 1922 reasoned that oxygen debt was directly dependent on the metabolic breakdown of this lactic acid. In 1933 Margaria (36) related its formation to work intensity, and others (5) had related the amount formed in a given exercise to the physical fitness of the subject. In 1934 Margaria (38) determined that the pay-off of lactic acid after exercise in a resting condition was an exponential function with a half time of fifteen minutes. By comparing this pay-off of lactic acid after exercise in a resting condition to the total oxygen debt he was able to determine that lactic acid was produced only in anaerobic exercise and that the conversion of pyruvate to lactate was not the only means by which the body derived

energy for muscular contraction under anaerobic conditions. Further work however, showed that the "alactic" processes only seemed to operate at the very beginning of exercise and that once the intensity of exercise had reached two thirds maximal for a particular person then lactic acid formation was the only anaerobic energy process.

In 1956-57, W. Huckabee surprised workers in this field when he published a series of papers (25,26,27,28,29) which presented evidence that there was no such thing as an "alactic" debt. He was able to explain the oxygen debt from the amount of excess lactate formed during exercise. As well, he said that lactic acid was formed during all exercise at a constant rate which was approximately equal to five percent of the metabolic rate. This was directly contrary to the findings of Edwards and Dill (13) who had published results which indicated that if a subject exercised at a very low intensity at the conclusion of an anaerobic exercise period, the lactic acid was oxidized at a considerably faster rate than if the subject had remained in a resting condition.

Since Huckabee's publications other investigators have found little evidence to support him. Some though, have agreed that a small quantity of lactic acid is formed at the beginning of any exercise (10,11,31,51,53,56). However, if the exercise is anaerobic their results indicate that the blood lactic acid levels reach a peak two to five minutes after exercise begins and then declines to almost

resting levels within ten minutes.

Statement of the problem

The purpose of this study was to determine how the time course of formation of lactic acid varies with the intensity of the exercise, and whether lactic acid could be metabolized during exercise.

Subsidiary problems investigated were:

1. The relationship of lactic acid formed over and above resting levels to the oxygen debt developed.

2. The rate of removal of lactic acid from the system in both exercise and rest conditions.

3. The concept that the "excess lactate" (which is that lactate theoretically formed solely because of oxygen lack and calculated according to a formulae developed by Huckabee) is a better measure of anaerobic metabolism than is a direct measure of increase in lactate.

Rationale behind the study.

From the findings of Edwards and Dill and some later investigators (10,11,51,53), one would expect that if exercise was begun at a rate calculated to be anaerobic, and then reduced to a low aerobic rate for a sufficient period of time, some of the lactic acid produced during the initial phase would be metabolized. However, one would not expect the level reached at the conclusion of the aerobic phase to be as low as it would be if the initial phase of exercise had been at an aerobic rate. A group of subjects were therefore subjected to three exercise treatments. Two were

similar to those just described except that at the end of the first two phases, a third short phase of almost maximal exercise was added. The third treatment consisted only of this short phase of almost maximal exercise. Lactic acid concentrations were followed throughout, and at the end of the exercise periods.

A comparison of results showing the time course of lactic acid formation in each treatment indicated whether lactic acid is built up continually during exercise (Huckabee) or whether it can be metabolized if the exercise is of low intensity. If Huckabee is correct, then one would expect a continual increase in lactic acid concentration throughout exercise, though with varying rate according to the level of the exercise and reaching its peak at the conclusion. If lactic acid can be metabolized however, one would expect a fall in concentration to occur, beginning as soon as the exercise level became aerobic.

Delimitations of the study.

1. The study is limited to ten university students of the University of Alberta.

2. Only the parameters stated in the problem will be considered.

Limitations.

1. The reliability of lactic acid formation for a given intensity of exercise for a given subject.

2. The magnitude of experimental error by the investigator.

3. The variation in humidity in the laboratory.

4. The ability of the investigator to motivate each subject to perform maximally in the test to determine their MVO_2.

5. The use of volunteer physical education graduate students restricts the randomness of the sample.

Definition of Terms.

1. Anaerobic work; that work performed without the simultaneous utilization of oxygen.

2. Aerobic work; work performed by means of energy derived only by oxidative metabolism.

3. Excess lactate, XL; Huckabee's term for lactic acid produced solely due to oxygen lack. He calculated it from the formula:

$$XL = (Ln - Lo) - (Pn - Po)(Lo/Po)$$

where Ln and Lo are experimental and control lactate concentrations and Pn and Po are experimental and control concentrations of pyruvate.

4. Maximal Oxygen Uptake, MVO_2; assuming pulmonary function to be normal, the maximal oxygen uptake is defined as the maximal rate of oxygen supply to and utilization by active tissue.

CHAPTER II

REVIEW OF THE LITERATURE

The relationship between lactic acid and oxygen debt.

Between 1920 and 1924 several investigators (23,32) found that after any form of exercise, a subject consumed more oxygen than he normally would at rest for a short time after the exercise had ceased. A.V. Hill (23) reasoned that this extra oxygen was being used by the body to replace a deficiency that had been caused as a result of the exercise. He therefore coined the term "oxygen debt" to describe what he regarded as a "paying back" of oxygen that had not been available at the time needed during exercise. An increase in the concentration of lactic acid in the blood had also been observed to occur as an effect of exercise.

As early as 1907 Fletcher and Hopkins (16) working with an isolated frog muscle, had demonstrated that lactic acid was produced when muscle contracts in the absence of oxygen, and that it accumulated with continued stimulation until the muscle became fatigued and could no longer contract. It was noticed that the increased concentration of lactic acid also decreased over what seemed to be a consistent time interval once exercise had finished. Combining these observations, Hill hypothesized that the oxygen debt was due to the oxidation of the lactic acid. He attributed the lactic acid build up to insufficient oxygen supply as compared to oxygen need during the exercise.

The formation of lactic acid during exercise. In 1933, Margaria, Edwards and Dill (38) published a paper which was a direct follow-up to Hill's earlier hypothesis, and which has become the classical study in this field. They found that an increase in the concentration of lactic acid in the blood only occurred after a subject had worked at an intensity which corresponded to at least two thirds maximal oxygen consumption. For most subjects this coincided with an oxygen uptake of approximately 2.5 litres per minute. This threshhold varied for different individuals according to their physiological condition. The better conditioned subjects could take a higher work load before an increase in lactic acid concentration occurred.

Therefore, oxygen debt could no longer be directly related to the oxidation of lactic acid. This type of oxygen debt which was formed without any increase in lactic acid concentration in the blood Margaria termed "alactic debt", or "alactacid debt". When increase in lactic acid concentration did occur, then the term "lactacid" was attributed to the portion of the oxygen debt needed to oxidize this lactic acid. The higher the work intensity, the greater the increase in blood lactate concentration and the greater was the accompanying oxygen debt.

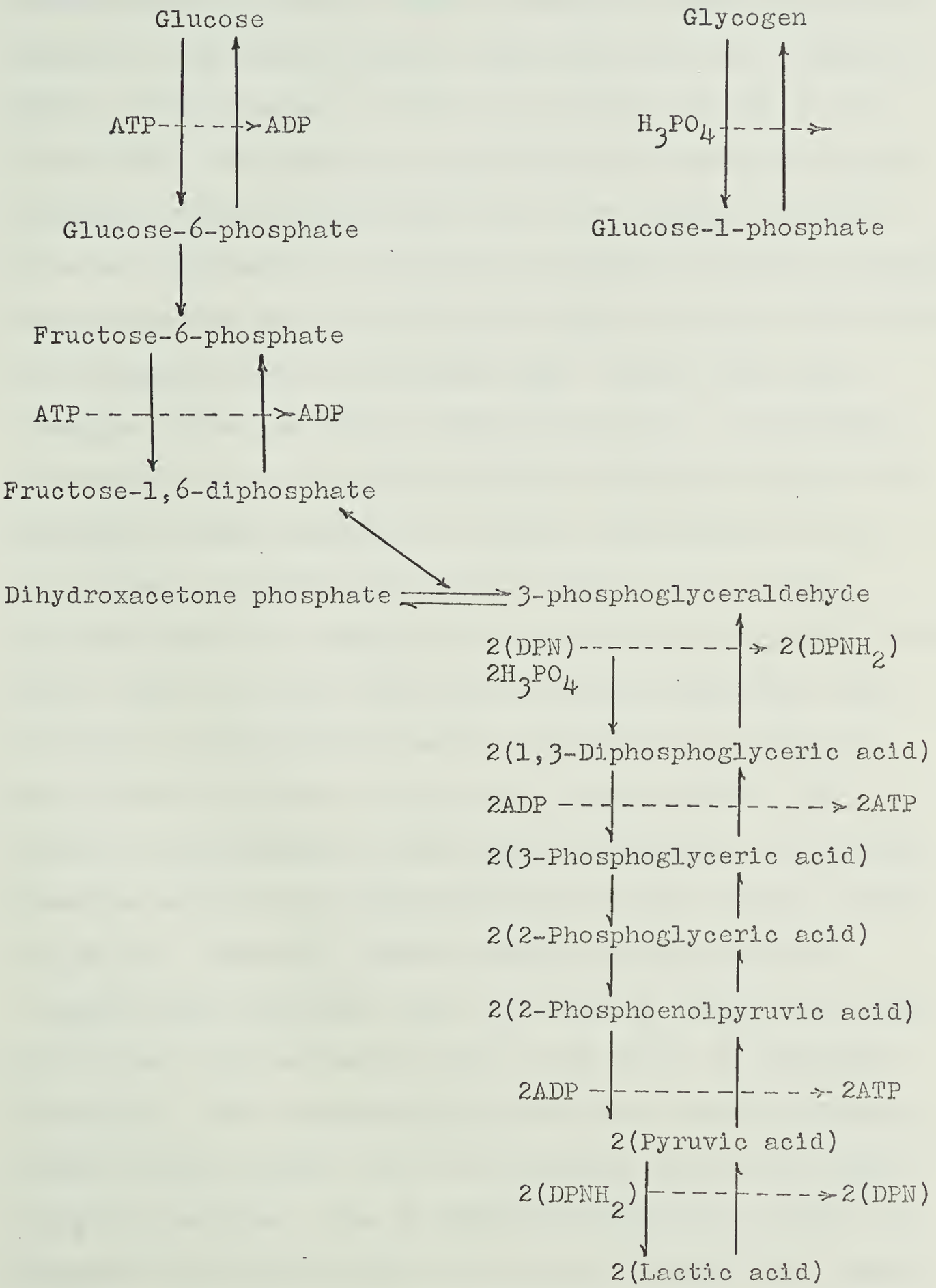
Margaria (40) explained this in the following way: "The significance of lactic acid production in exercise is an emergency mechanism for providing energy when the oxidative mechanism is insufficient."

These statements indicate that lactic acid production occurs when the oxygen transport system of the body cannot provide sufficient oxygen for the body's needs. This is explained as follows. Adenosine-triphosphate (ATP) is the immediate source of energy for muscular contraction. ATP can be produced in two ways. Usually oxygen is available and it is then produced through the aerobic pathway. Fats proteins and carbohydrates can be source material in the production of energy aerobically, as they enter directly into the Krebs cycle. Anaerobically however, only carbohydrate can be the source material. It is metabolized via enzymatic reactions in the phospho-glyconic pathway (17). In both cases (aerobically and anaerobically) it is converted to pyruvic acid. Under aerobic conditions this then enters the Krebs cycle. In anaerobic conditions, to enable the ATP forming reactions to continue, pyruvic acid does not enter the Krebs cycle, but is converted to lactic acid with the aid of the reduced coenzyme Nicotinamide adenine dinucleotide (NADH)(55).

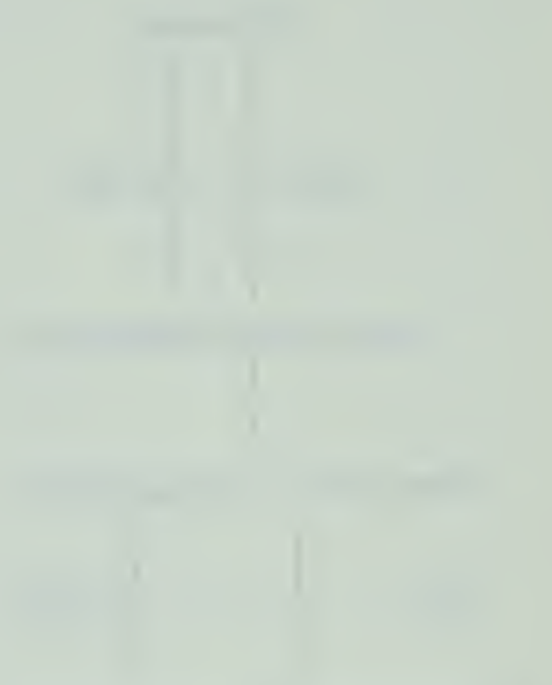
$$\text{Pyruvic acid} + NADH_2 \xrightarrow{\text{lactic dehydrogenase}} \text{Lactic acid} + NAD + 2H$$

i.e. lactic acid serves as a H^+ acceptor in the production of NAD^+ which is required to enable the phosphoglyceric pathway to continue functioning. (NAD is converted to NADH in the reaction 3-phosphoglyceraldehyde to 1,3-diphosphoglyceric acid). Therefore, in the total glycolytic chain lactate formed anaerobically has the function of being an acceptor of hydrogen electrons in the oxidation of NADH.

THE GLYCOLYTIC PATHWAY



Summary of reactions between glucose and lactic acid.



Observed lactic acid concentration in exercise. The concentration of lactic acid in arterial blood of the normal human at rest varies between approximately 0.6 to 1.5mM per litre of blood water, or from 5 to 13 mgm per 100 cc of blood (25). Following an anaerobic run to exhaustion on a treadmill at 18.7 km per hour on a five percent grade for four and one half to six minutes, Margaria obtained a concentration at the end of exercise of 17mM per litre of bloodwater or 120 mgm per 100 cc of blood (38). Others (11) have obtained values as high as 328 mgm percent. By plotting recovery lactic acid values against calculated oxygen debt values it seemed apparent to earlier investigators that no increase in lactic acid concentration in the blood occurred until the oxygen debt amounted to about three litres. After this, i.e. once the work had become anaerobic, the increase in blood lactic acid concentration was seen to be a linear function of the amount of work (37). This in turn is proportional to the oxygen consumption. Margaria's results seemed to be substantiated by other studies (13,17, 20,39,59). However, several studies (13,49) produced results which indicated that there was an increase in blood lactic acid concentration even at the start of submaximal exercise. This subsequently returned to a level slightly above resting values two to four minutes after the beginning of exercise. Then W. Huckabee published a series of reports (25,26,27,28,29), the results of which were almost the complete reverse of Margarias. Since Margaria's work

the function of NAD and NADH and the lactate dehydrogenase system in the glycolytic pathway and their relationship to the ratio of lactate to pyruvate present had been established. On the basis of this, Huckabee queried the validity of equating lactic acid formation with oxygen lack by showing that lactic acid could be produced under other circumstances. His results showed that he had produced blood lactate increase by having subjects hyperventilate, by infusing sodium bicarbonate and by infusing pyruvate. As a result of this he developed a formula based on the lactate dehydrogenase system from which could be calculated the proportion of hypoxic lactic acid from the total amount of lactic acid formed during exercise. This amount he termed "excess lactate". He theorized that the ratio lactate/pyruvate represented in effect the ratio NAD/NADH and therefore that any change in this ratio in excess of what could be attributed to change in pyruvate present could be assumed to be caused by oxygen deficiency. To calculate the excess lactate his formula was of the form:

$$XL = (Ln - Lo) - (Pn - Po)(Lo/Po)$$

Most important from the point of view of this study was his finding of excess lactate at low levels of work with small oxygen debts. Further, when he calculated the quantity of oxygen necessary to convert the peak excess lactate back to pyruvate he found close approximation to the measured oxygen debt. He also stated that the amount of excess lactate present increased linearly with time at a rate approximately

equal to five percent of the metabolic rate at all work levels. Therefore, Huckabee denied the existence of the alactic portions of the oxygen debt and stated that all of the debt could be explained at all levels of work by the formation of excess lactate.

The results of a paper published soon after Huckabees (7) did show similar increases in blood lactate levels during all exercise. However, the subjects used in the study were patients with arteriosclerosis. It may therefore have been impossible for them to work aerobically. Certainly their highest blood lactate concentrations were obtained at a much lower load and pulse rate than in normal persons.

Since that time several studies have been made, (10,19, 31,51,52,53,56) but few of the published results support Huckabee's findings. Olson (44) strongly criticized Huckabee's work, citing several false premises. The first was that Huckabee had assumed that in open systems typical of mammalian cells it was equilibrium constants that determined the steady state concentrations of substrates in the system. Rather, says Olson, it is the rate of the various competing reactions that is the determining factor. The second false premise was that the lactate/pyruvate ratio accurately reflects the NAD/NADH ratio in the aggregate of cells under study. This may not be so for several reasons. Firstly, there are at least two pools of NAD/NADH in the cells, one in the cytoplasm and one in the mitochondria. Secondly, these pools do not participate in direct trans-

hydrogenation, but rather a shunt metabolite shuttles hydrogen ions between the cytoplasm and the mitochondria. Therefore Olsen states that "while excess lactate is not an indisputable sign of anaerobiosis, and conversely that anaerobiosis can occur without the appearance of excess lactate, it is equally true that simple anoxic anoxia causes the accumulation of excess lactate."

H.G. Knuttgen (38) in a study to determine the effect of activity of large muscles at different work intensities on oxygen debt and on lactate, pyruvate and excess lactate during work and recovery used two subjects on a bicycle ergometer at work intensities of 300, 700, 1,000 and 1600 kgm per minute. He found that no appreciable rise in excess lactate and total lactate occurred at the two lower intensities of work. For his subjects the critical level of oxygen uptake after which there was a rapid rise in total lactate and excess lactate was 1.5 litres per minute. At all levels of work, the oxygen debt he found surpasses in quantity the oxygen equivalents of the maximum increases in total lactate, and "to an even greater extent, the oxygen equivalents of excess lactate." This work therefore supported Margaria's findings of a separate alactacid and lactacid portion of oxygen debt.

H.D. Thomas et.al. (53) used both patients with normal and subnormal cardiac output and determined lactate, pyruvate and excess lactate formation. The work load varied for each subject, ranging from 266 to 930 kgm per minute. He found

that the rate of both lactate and excess lactate accumulation was a variable, and not a constant as reported by Huckabee. At these work rates, he found that the highest concentrations of blood lactate occurred at approximately two minutes of work after which it began a slow decline. "In many of the subjects, both normal and abnormal, the rate of excessive lactate accumulation was actually negative during the last minute of exercise, reflecting a decrease in the lactate/pyruvate ratio."

Harris, Bateman and Gloster (19) published similar findings. Cobb and Johnson (10) using a period of prolonged exercise in a group of sedentary normal individuals and in a group of physically active normal persons, likewise found a rapid accumulation of excess lactate during the first few minutes of exercise which then decreased in both groups. The physically active group showed a significant decrease in the excess lactate concentration after two to five minutes of steady exercise, to a plateau just above resting level. The level in the sedentary subjects continued to increase, but at a much reduced rate.

Other studies found essentially identical results. Wasserman (56) concluded that the anaerobic metabolic rate, whether calculated from excess lactate or oxygen debt was not a constant fraction of the metabolic rate at all work loads. "A larger fraction of energy is derived from oxygen debt creditors at heavy and very heavy than at moderate work loads." Saiki's results (51) showed that at 70-80 percent maximal oxygen consumption, lactic acid is produced

only at the onset of exercise during the oxygen debt contraction phase. He found no lactic acid produced once steady state oxygen consumption was reached. However, at 80-100 percent maximal oxygen consumption "lactic acid concentration in the blood increases earlier and arrives at a maximum somewhat later than at seventy percent of maximum aerobic capacity."

Schneider (52), whose results were almost identical to those of Saiki, proposed that the lag in oxygen consumption at the start of aerobic exercise was more directly dependent on the time required to develop an oxygen need, and thus prepare tissues to accept oxygen, than on a lag in the oxygen supplied by the circulatory and respiratory system. The need is developed by the formation of lactic acid at the start of exercise. Henry had proposed this in 1951 (18) and Margaria in 1965 (37). It seems that apart from the investigation of Edwards and Dill in 1936 there has been no attempt to follow the time course of lactic acid concentration having first elevated it to a high level by having a subject perform anaerobic work and then reducing the level of work to a submaximal one. If, as the investigators just cited submit, lactic acid formed in the early part of aerobic exercise is removed if the exercise continues at an aerobic level, one would expect that a higher lactic acid concentration purposely formed by anaerobic exercise could likewise be reduced if the level of exercise was reduced to become aerobic.

Removal of excess blood lactate concentrations. Reconversion to pyruvate is not the only method used by the body to remove excess lactic acid, though it is the most important. Thus, the lactacid portion of an oxygen debt is used to convert lactic acid back to glycogen. Some may be lost through sweat and urine, though it has been estimated that this would be only one percent. The heart uses lactic acid for fuel and ten percent may be removed in this manner (13).

The limiting factor in the removal of lactic acid is probably the speed with which it can be relayed to the reactive centres, e.g. liver, skeletal musculature and heart. This would depend on circulatory rate.

Margaria (37) determined that the rate of removal from the blood of the extra lactic acid formed in exercise was an exponential process with a half reaction time of fifteen minutes. The rate of removal is therefore proportional to the amount of lactic acid present.

However, it should be noted that the above rates of removal apply from the time when the lactic acid concentrationis at its maximum. Following maximal exercise this point is generally not reached until six to eight minutes after the completion of the exercise.

Since rate of removal appeared to be limited by the limitation imposed by circulation in getting the lactic acid to the reactive centres, Edwards and Dill (13) designed a study to determine what would happen if during recovery, work was performed at levels of oxygen consumption which

do not raise the lactic acid level. They varied this rate of work up to twelve times the basal rate of oxygen consumptionfor forty five minutes. They found that lactic acid was removed faster than at rest--that "the rate of removal of lactic acid in exercise increases approximately proportionately with the metabolic rate up to some critical level of activity, different for each subject." For one subject, a trained runner, the velocity constant was increased 3.5 times the value at rest. No further work appears to have been done as a follow-up to this, possibly because some other investigators found an increase in blood lactic acid concentration in all exercise. However, as described above, recent studies have shown that following an initial rise in lactate at the start of aerobic work, the lactate concentration then declines to almost resting values. From the data presented, it appears to be a relatively rapid decline, though no comparative values have been calculated. As Saiki states, (51) this indicates "that the oxygen consumption in equilibrium is sufficient not only to supply the energy for the actual work performed, but also to provide for the energy necessary to reconvert lactic acid to glycogen."

Physical fitness and lactic acid formation. Physical fitness in this context refers to physical fitness as determined by maximal oxygen intake. There is a negative correlation between physical fitness and the build up of excess lactic acid in exercise(5,6,10,17,45,49,53). Correspondingly, less fit subjects have a lower critical intensity of exercise beyond which increases in blood lactate appear. Conversely

the fitter subjects have a greater tolerance to lactic acid (49). That is, they can derive a greater proportion of their energy requirements anaerobically.

Determination of blood lactate concentration. Blood lactic acid is an index of the total lactic acid produced as a result of exercise (38). Margaria found no difference in lactic acid values obtained between femoral or arm samples of arterial and venous blood. It has been shown that lactic acid diffuses rapidly from tissue cells producing it into blood plasma and from there to less active muscle (12). However, Huckabee states that he obtained large significant differences in results when he used peripheral venous blood samples as opposed to arterial or mixed venous blood samples. This was presumably because the peripheral samples would reflect local events in the limb rather than total body lactate changes. Thus, despite Margarias findings most later researchers have attempted to control possible error source as much as possible by taking samples from the brachial artery.

CHAPTER III

METHODS AND PROCEDURES

Ten male graduate students enrolled at the University of Alberta in the faculty of physical education volunteered as subjects for the study. The same subjects were tested under the three experimental conditions.

In the first week of testing, each subject reported once. Anthropometric data was collected, the subjects were informed about the nature of the experiment and their maximum oxygen uptake was ascertained. From this, aerobic and anaerobic work loads were calculated for the testing sessions to follow.

In the following six weeks each subject was tested on each of the three experimental conditions described below. Trials were counterbalanced. Testing was always conducted between 1 p.m. and 4 p.m. in the afternoon. Subjects were asked to maintain their normal day to day habits as much as possible throughout the testing period and not to engage in any strenuous activity on the morning of the test. Temperature in the laboratory ranged from 21 degrees centigrade to 23 degrees centigrade during all testing.

Experimental apparatus.

1. Bicycle ergometer. A Monark GCI bicycle ergometer was used to provide the work load for the subject. A bicycle ergometer was chosen because it provided a convenient method for administering a direct test for maximal

oxygen uptake, because work loads were relatively easy to set and adjust through the exercise periods and because the subject's arms were fixed and samples of blood could be taken from the brachial artery without the subject having to stop work. The bicycle was calibrated prior to any testing.

2. Gas collection and analysis. An Otis Mckerrow two way breathing valve was used which enabled the subject to inhale room air and expire into a Douglas bag which was connected by means of a 1.5 inch diameter hose. A nose clamp was used to prevent respiration through the nasal passages.

Oxygen analysis involved use of a Beckman model E-2 oxygen analyser. A 60 cycle Neptune Dyna-pump, model 2, was used to pump a gas sample through the analyser. Two readings were taken, and if no change in value was noted the value was recorded. For each 15 second reading, 75 cc of air was added to the total volume of the Douglas bag sample.

A Godard Capnograph was used for Carbon dioxide analysis. Gas Volume was measured in litres with an American volume meter (model 802).

3. Lactate analysis. Blood drawn for each sample was immediately mixed with 5 ml TCA in a test tube. Samples were stored in a refrigerator until approximately thirty had been collected. They were then analyzed spectrophotometrically using the enzymatic method of Ellis, Cain and Williams (15). A Unicam SP 800B spectrophotometer was used. Lactic acid is converted to pyruvic acid by lactic

dehydrogenase in alkaline solution by using excess DPN and the pyruvate is removed as it is formed by hydrazine. The DPNH formed is read at 340 mu and gives a quantitative measure of the lactic acid originally present.

4. Pyruvate analysis. Samples were analyzed spectrophotometrically using the chemical method of O'brien and Ibbott (Appendix A).

Experimental procedure.

1. Determination of aerobic and anaerobic work rates. The test for maximum work capacity was carried out using the Astrand-modified Physical Work Capacity test(3). The subject warmed up for six minutes at a 600kpm work level, at fifty revolutions per minute. This was followed by a five minute rest interval. The work load was then increased to 900kpm and the subject repeated the procedure. At the end of four minutes and thirty seconds of the ride an Otis McKerrow two way breathing valve was connected to the subject by means of a rubber mouthpiece and his nose was completely closed with a nasal clamp. An expired gas sample was then collected in a Douglas bag between the fifth and sixth minute of the ride. For the next five minutes the subject rested while the work load was increased to 1200kpm. At this load and above, the work time was reduced to four and a half minutes, gas being collected in the three and a half minutes to four and a half minute time period. This procedure was repeated until oxygen uptake levelled off or declined, this value being taken as the subject's maximal oxygen uptake. The values of 30 percent, 50percent and 90 percent used in the following

experiments were calculated from this value (Appendix C). According to previous investigators, the first two values should represent aerobic work loads, and the last an anaerobic work load (28,31,36,37,51).

2. Collection of blood samples. The procedure used in testing under each experimental condition was the same. The subject reported, and rested for approximately 10 minutes. A three minute gas sample was then collected and analysed to determine resting oxygen consumption.

The subject remained in a lying position and a Cournand needle was inserted into the brachial artery. A local anaesthetic was used to lessen pain, and once in position the needle was heavily taped. To help prevent it from slipping out of the artery in the longer trials the subject's arms were held straight and taped to a splint.

A resting 6-8 cc blood sample was then drawn. From this, blood density, blood water, and resting lactate and pyruvate concentrations were later determined. Following this the subject mounted the bicycle which was adjusted to his leg length. The work load was adjusted as soon as the subject began to pedal, and was regularly checked throughout the procedure. Blood samples of approximately 3 cc were draen at 5, 10 and 14 minutes after beginning work for the two longer trials ((a) and (b) below). The rates of work used in the three experimental conditions were:

(a). Ninety percent maximal oxygen uptake for one and a half minutes followed immediately by;

Thirty percent maximal oxygen uptake for fifteen minutes, followed by;

Ninety percent maximal oxygen uptake for ninety seconds.

(b). Fifty five percent maximal oxygen uptake for one and a half minutes followed by;

Thirty percent maximal oxygen uptake for fifteen minutes, followed by;

Ninety percent maximal oxygen uptake for ninety seconds.

(c). Ninety percent maximal oxygen uptake for ninety seconds.

In the last one minute of exercise gas collection apparatus was fitted to the subject and gas was collected from immediately the subject ceased work for twenty minutes. Upon ceasing exercise the subject remained seated on the bicycle. Recovery blood samples were drawn at 2,5, and 8 minutes from the end of exercise, and the gas was collected in Douglas bags which coincided with these time periods. Lactic acid and oxygen debt could thus be compared to determine whether lactic acid could in fact account for all of the debt.

3. Determination of blood water. Two ccs of blood from each resting sample were pipetted into beakers and weighed on a chemical balance. The samples were then transferred to a drying oven where they remained at a temperature of 93-95 degrees centigrade for two days. The beakers were then removed from the oven, allowed to cool in a dry air

atmosphere and reweighed. The difference in weight represented the weight of water/2cc blood.

4. Determination of blood density. A one cc pipette was first weighed and exactly one cc of blood then drawn up into it. The pipette was then reweighed to determine the weight of the blood. The density could then be calculated from the formula Volume = Weight x Density.

5. Calculation of oxygen equivalents. The method used to calcualte the oxygen equivalents of lactate and excess lactate was that described by Huckabee (25). Since the raw data was obtained in mgm/100 ml of arterial blood it is necessary to calculate from this the amount of lactate or excess lactate present in the entire body in order to determine from this the total oxygen debt. This in turn means that several assumptions are made. Firstly, it is assumed that the lactate values in millimoles/litre of blood water obtained from blood taken from the brachial artery is representative of tissue changes over the greater portion of the body. There seems to be sufficient evidence that this is so (25). Secondly, it has been assumed for calculation in this study that Total body water can be predicted from a knowledge of body weight. Again, the formula used appears to give an accurate prediction for normal persons (41). It is:

$$\text{Total body water (litres)} = (79.45 \times \text{body weight}/100) - ((0.24 \times (\text{body weight})^2)/100) - (0.15 \times \text{age} \times \text{body weight}/100).$$

Therefore, a figure can be obtained expressing total lactate and excess lactate values in millimoles for any one subject.

The oxygen equivalence of this figure is then obtained by multiplying it by its oxygen equivalence factor of 11.2 ml per mM lactate, thus expressing oxygen debt and body lactate in the same units for comparison.

CHAPTER IV

RESULTS AND DISCUSSION

The main purpose of this study was to determine whether a high level of lactic acid formed in the body could be reduced while exercise was still being performed, but at an aerobic level. If it could be removed it would indicate that lactic acid is not formed at all levels of exercise at a rate which is a constant and proportional to approximately five percent of the metabolic rate. It would also indicate that exercise has to be anaerobic in nature before lactic acid levels rise.

Raw data obtained from the spectrophotometer was converted to lactate and pyruvate values (all in mM/litre of body water), increase in lactate (ΔL), excess lactate, oxygen equivalent of excess lactate and the oxygen equivalent of increased lactate by an IBM 360 computer using a FORTRAN program compiled by the investigator. This program is contained in Appendix 'A'. Methods and formulas used in this program are also contained in Appendix 'A' and in the chapter entitled "Methods and Procedures".

Figures I to IV on the following pages represent graphically the results obtained for each subject on each of the three treatments. The figures for each point plotted are contained in Appendix 'B'. In each case values for ΔL are plotted on the ordinate against time at which the sample was taken on the abcissa. Figure I and figure III represent

the average values for ΔL and XL respectively for each treatment for the ten subjects.

Consider first the individual graphs for ΔL (figure II). In the case of treatment I, the 90 percent, 30 percent, 90 percent MVO_2 condition, ΔL, rises abruptly in the first one and a half minutes of exercise in every case. Since the 90 percent MVO2 level is well into anaerobic exercise this is to be expected from the findings of all previous investigators. During the following fifteen minute period however, when the exercise level has been reduced to the aerobic 30 percent MVO_2, ΔL decreases in the case of every subject, reaching almost resting levels in nine subjects and in the case of one subject (number 9) even falling slightly below the measured resting level. Thus it becomes apparent that if ΔL is used as a measure the increased lactic acid formed during a short, but intense exercise bout can be metabolized while performing aerobic exercise, bringing the body lactate level back to approximately resting values. In the final one and a half minutes of exercise at 90 percent MVO_2 ΔL values again rise abruptly as expected and in only three of the ten subjects did the peak values exceed the peak value of the initial 90 percent bout and then only by .3, .2 and .2 mMs./ This will be mentioned further in the discussion. The same treatment graphed for XL might be expected to show different results if the formation of the increased lactate is being influenced by factors other than hypoxia (25).

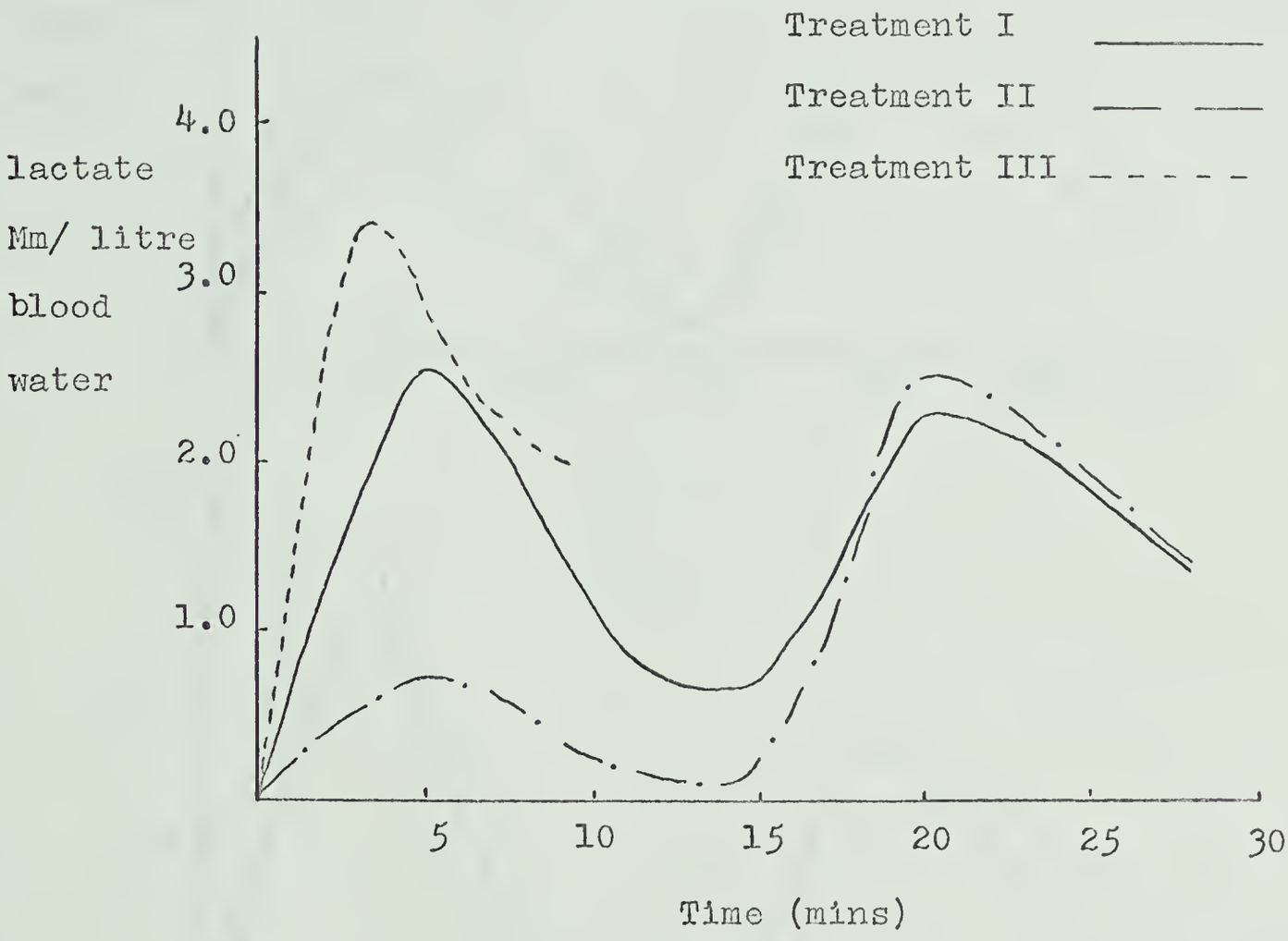


FIGURE I Graphs showing the mean values for ΔL for all treatments.

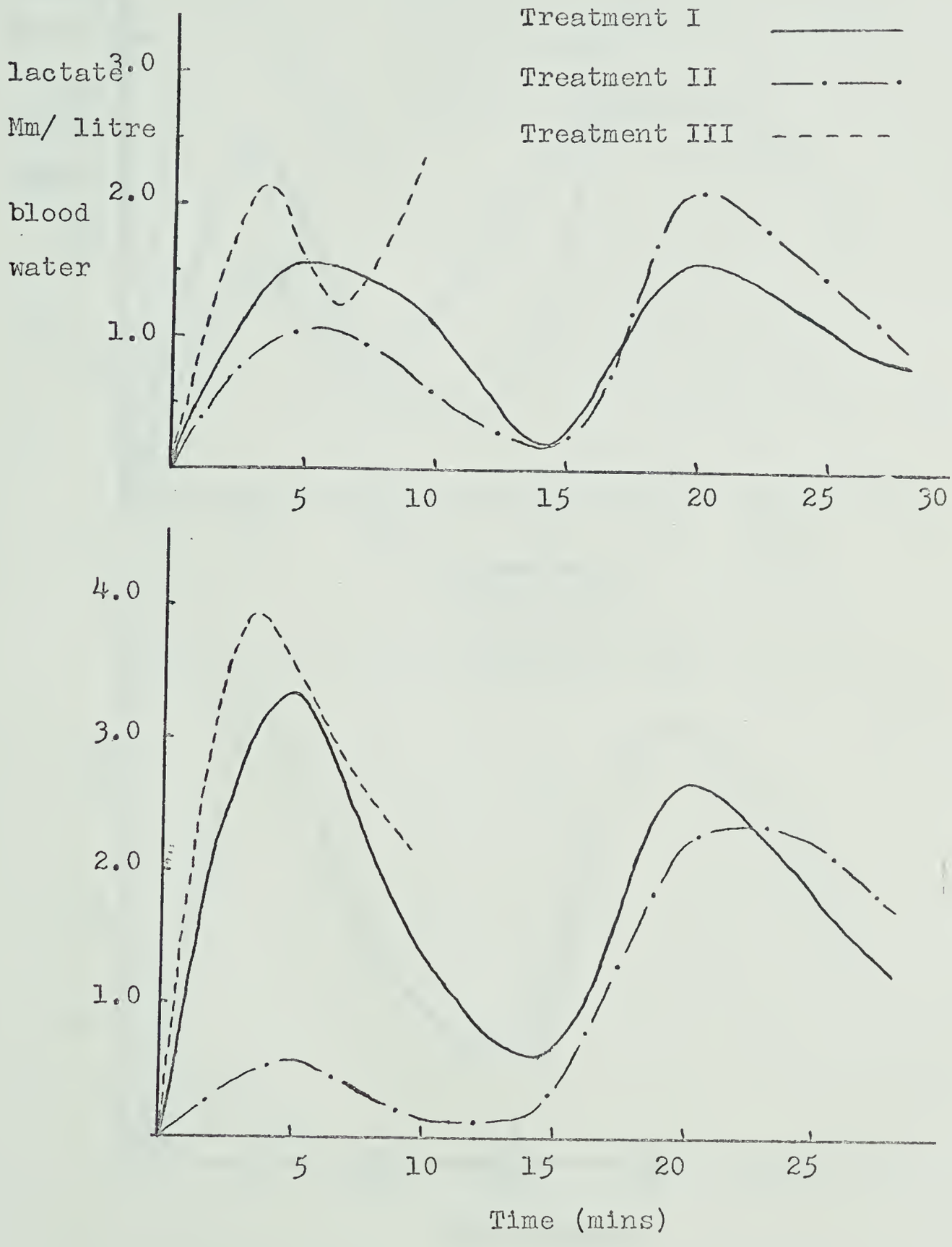


FIGURE II: 1 Graphs showing ΔL values for all treatments for subjects 1 (top) and 2 (bottom).

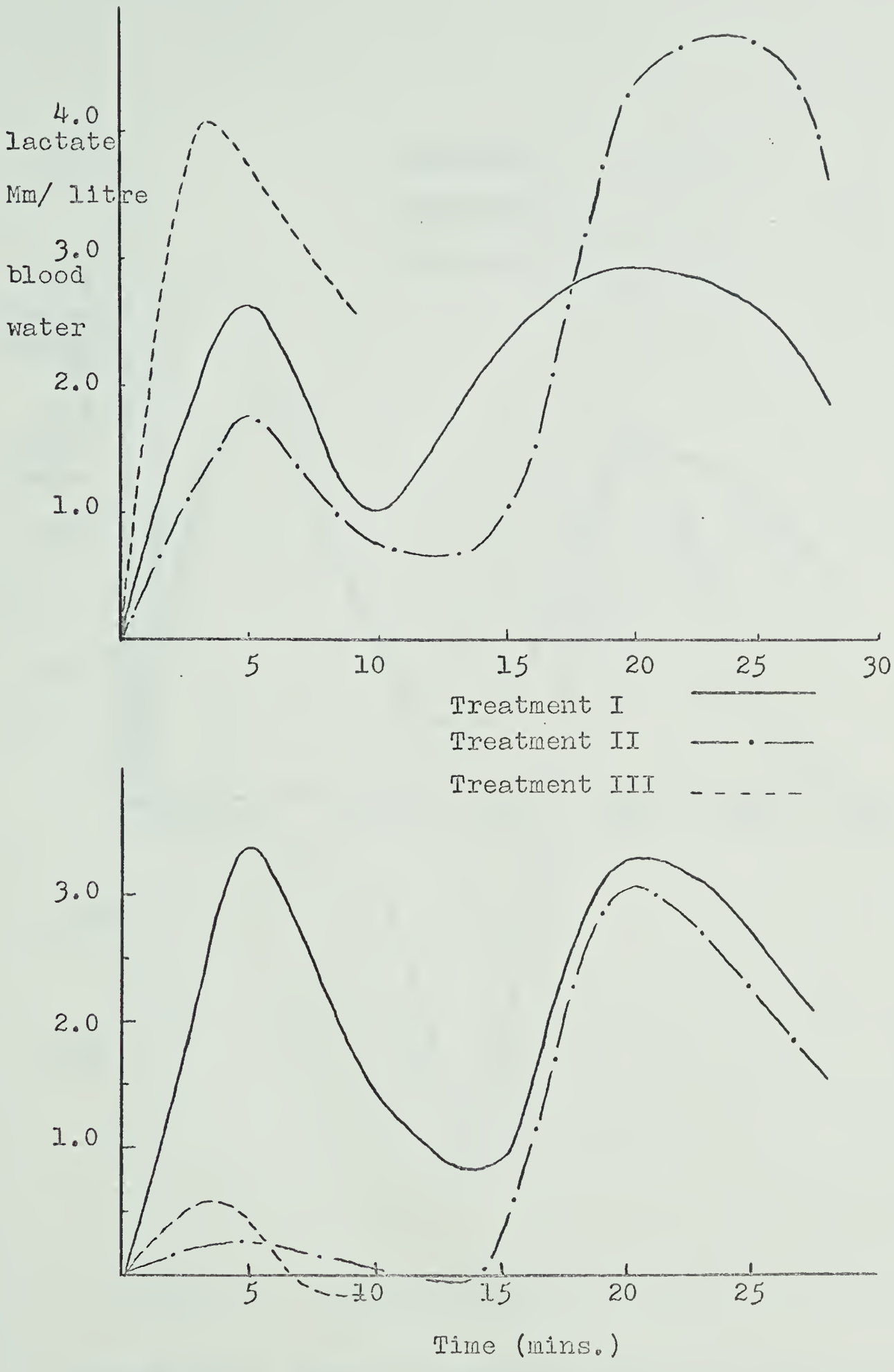


FIGURE II: 2 Graphs showing ΔL values for all treatments for subjects 3 (top) and 4 (bottom).

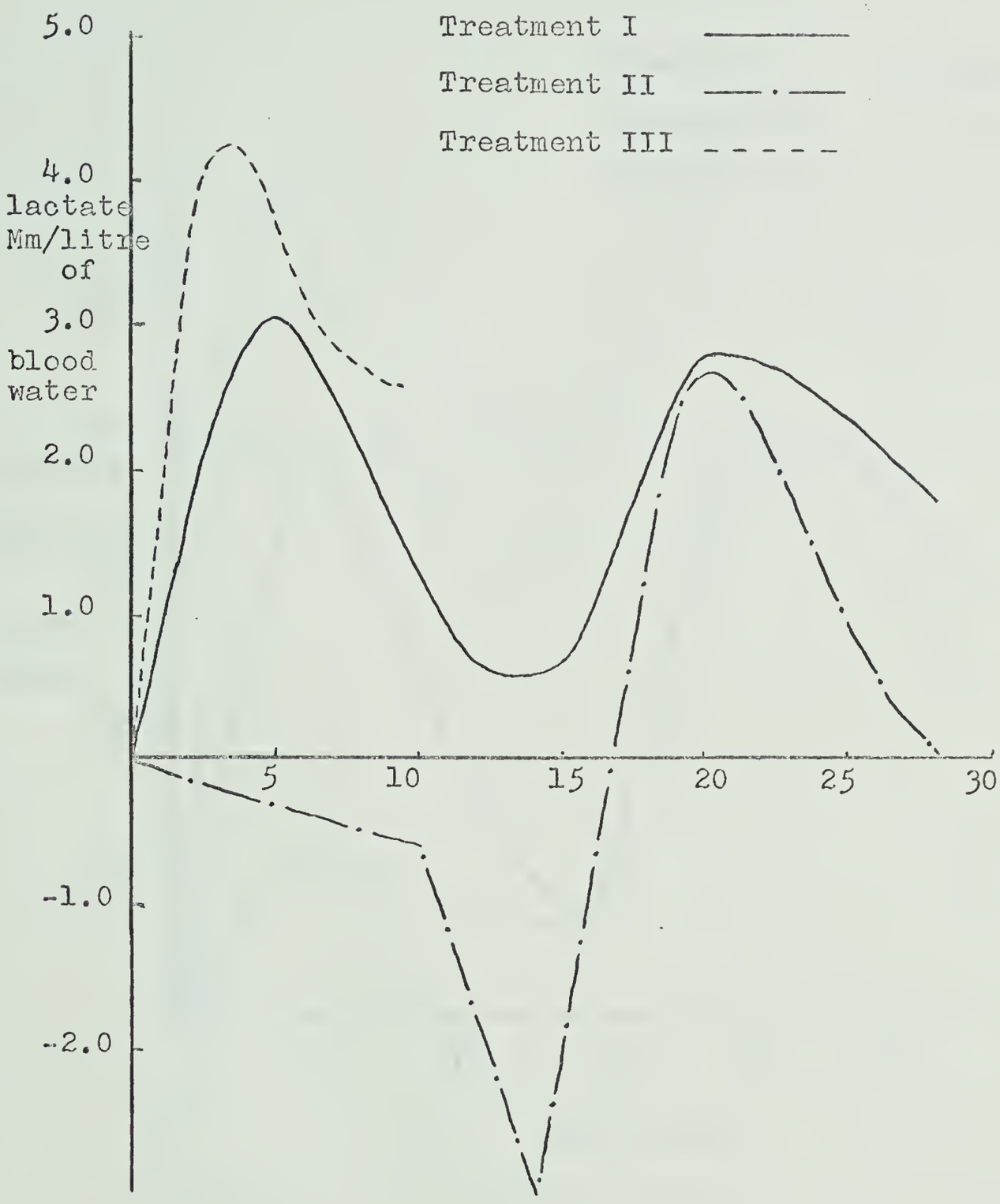


FIGURE II: 3 Graphs showing ΔL values for all treatments for subject 5.

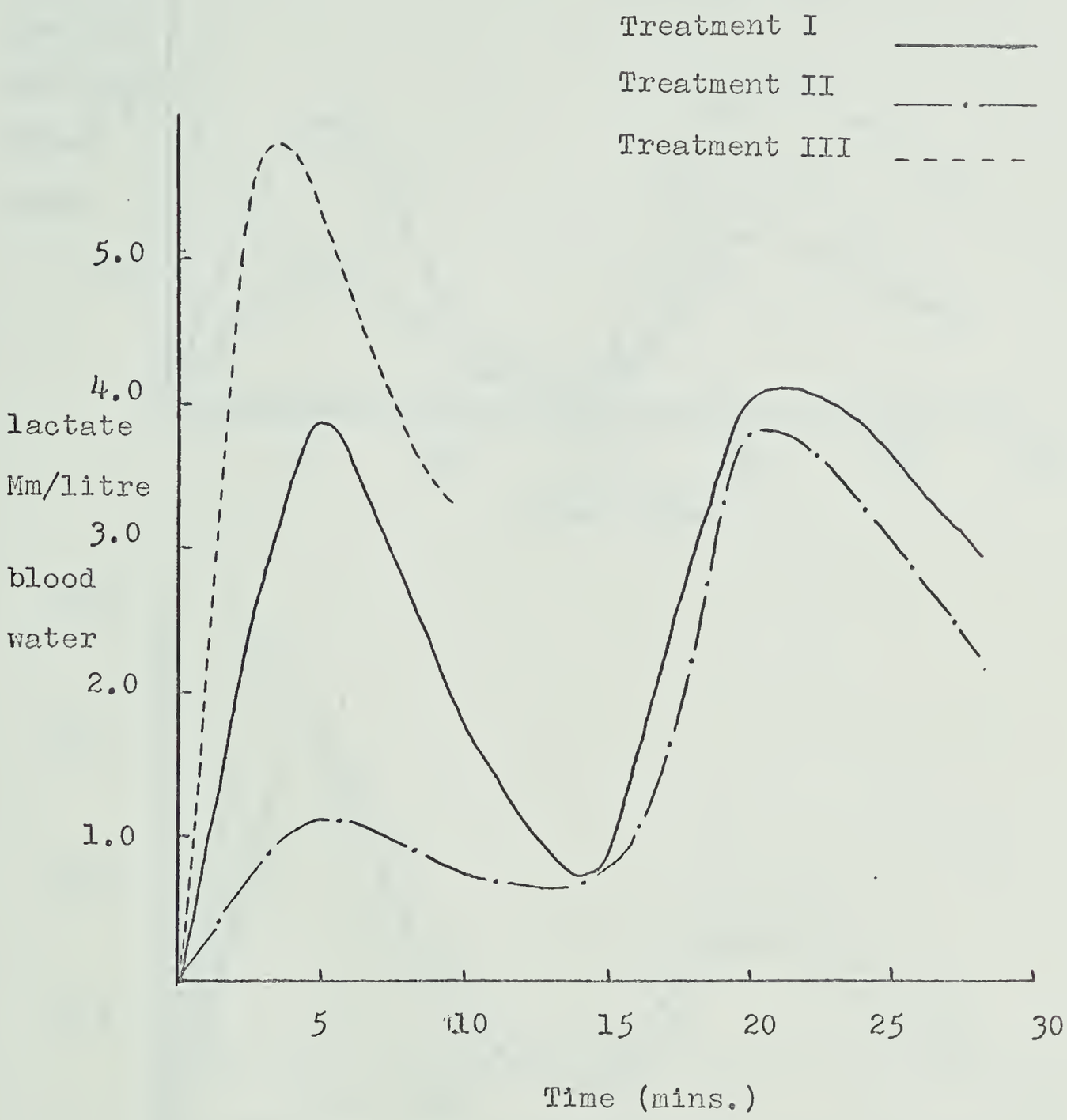
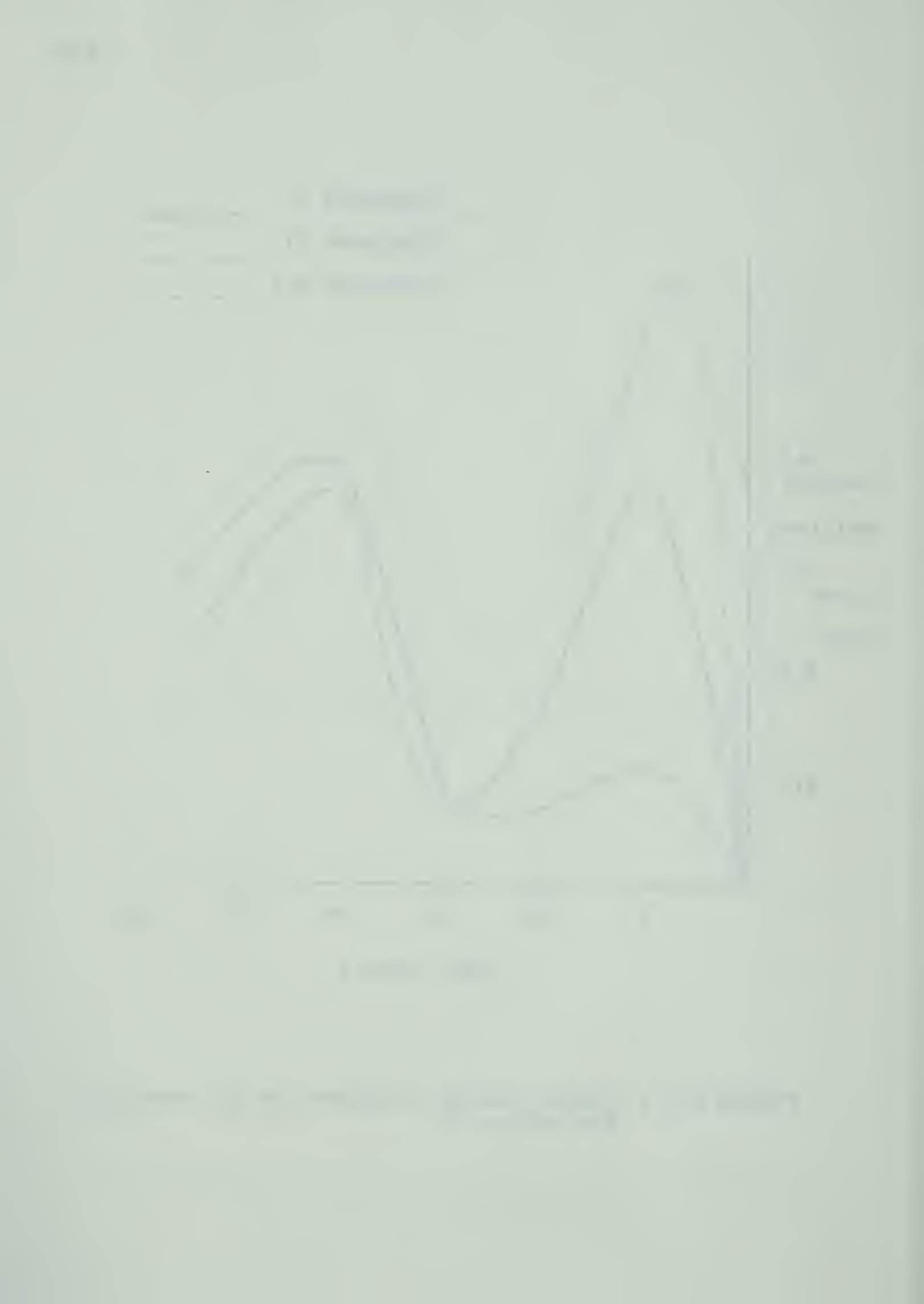
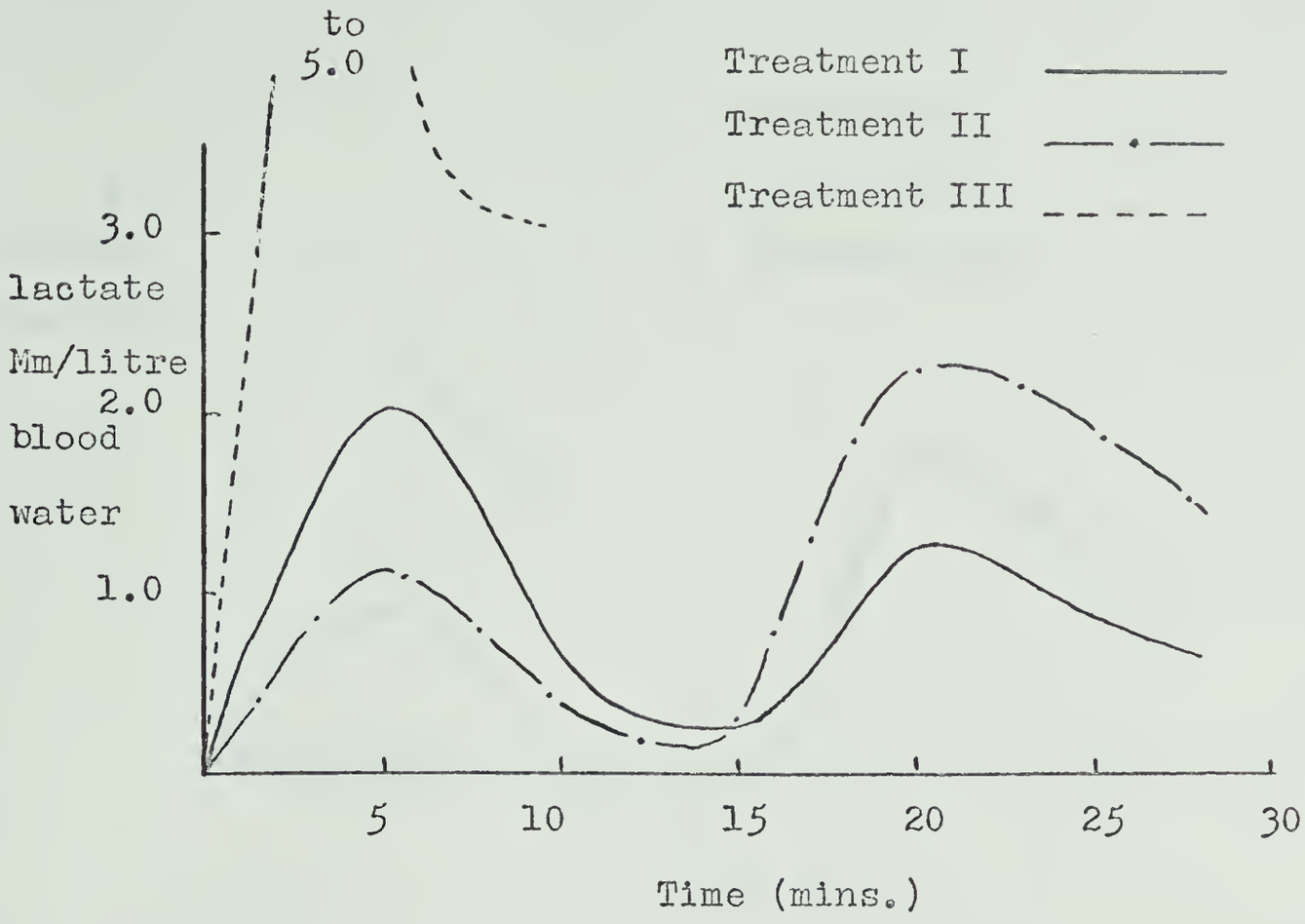


FIGURE II: 3 Graphs showing ΔL values for all treatments for subject 6.





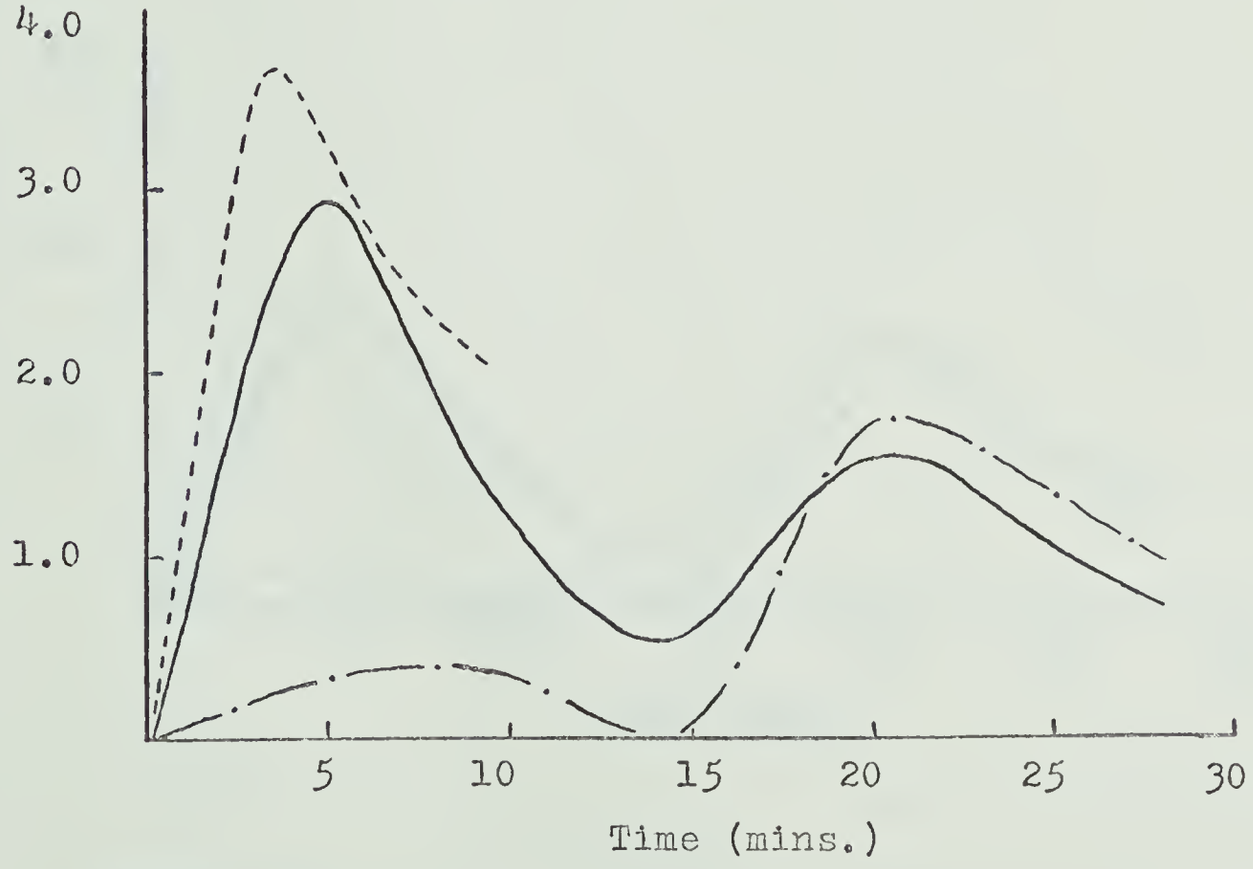
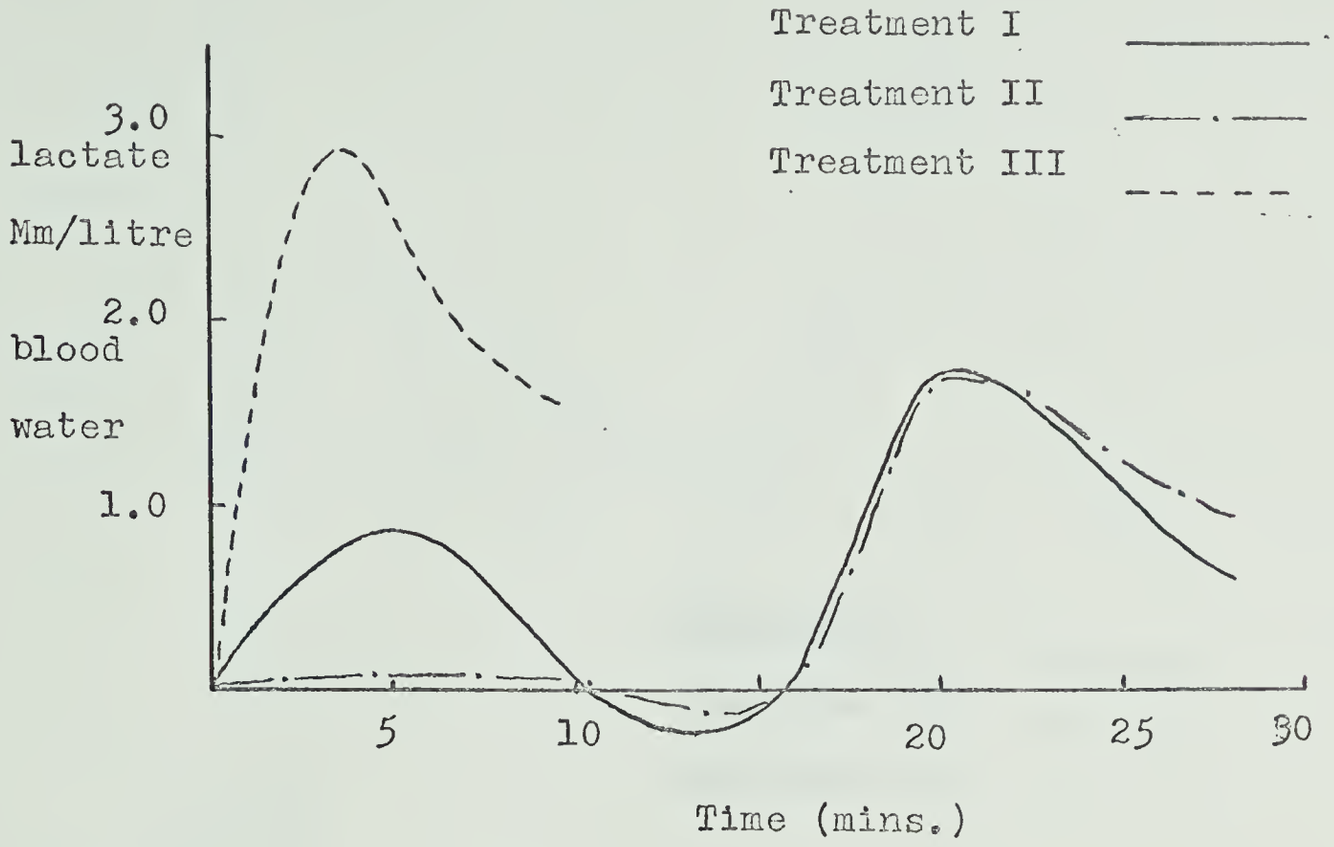


FIGURE II: 4 Graphs showing AL values for all treatments for subjects 7 (top) and 8 (bottom).



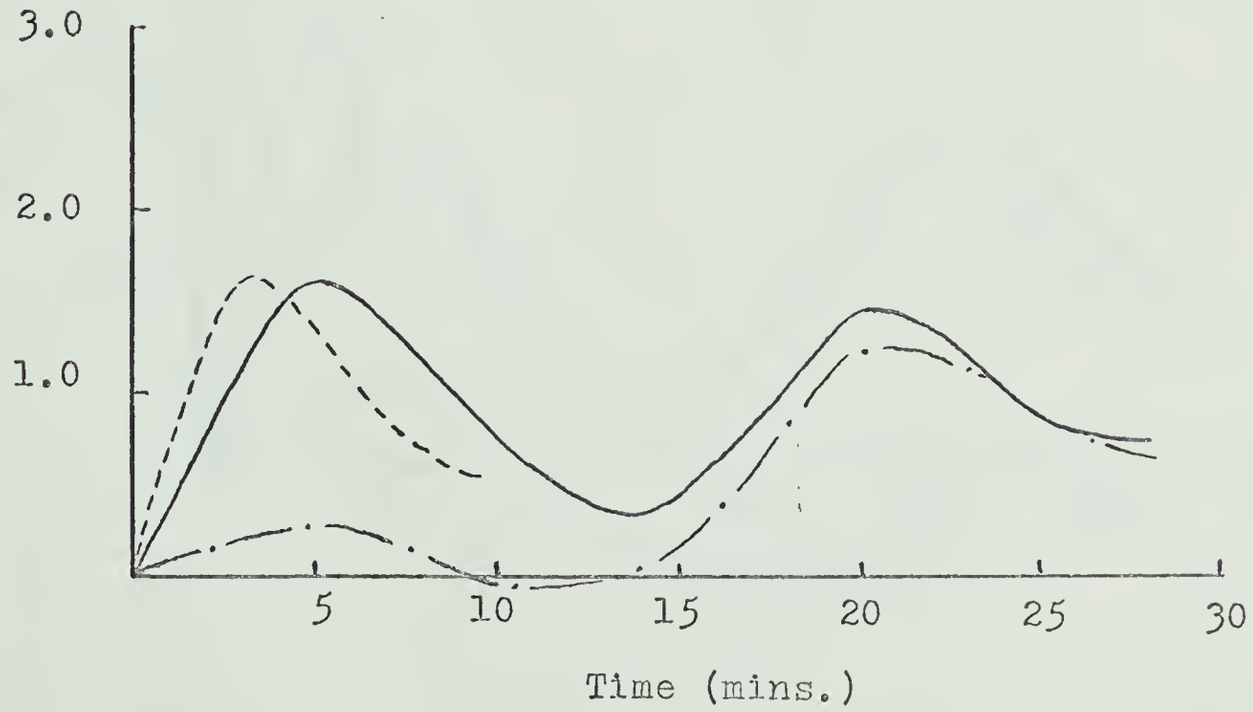


FIGURE II:5 Graphs showing ΔL values for all treatments for subjects 9 (top) and 10 (bottom).

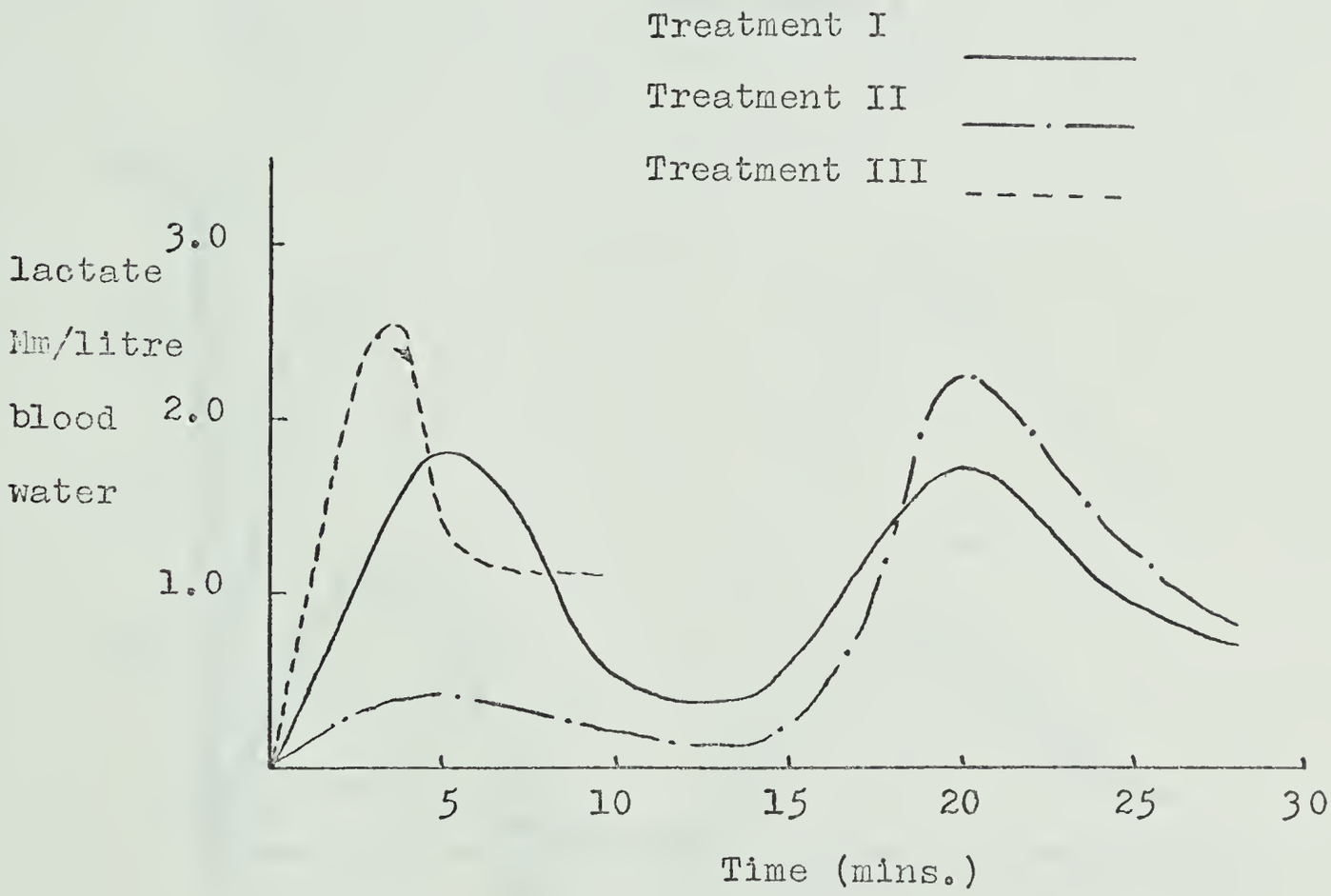
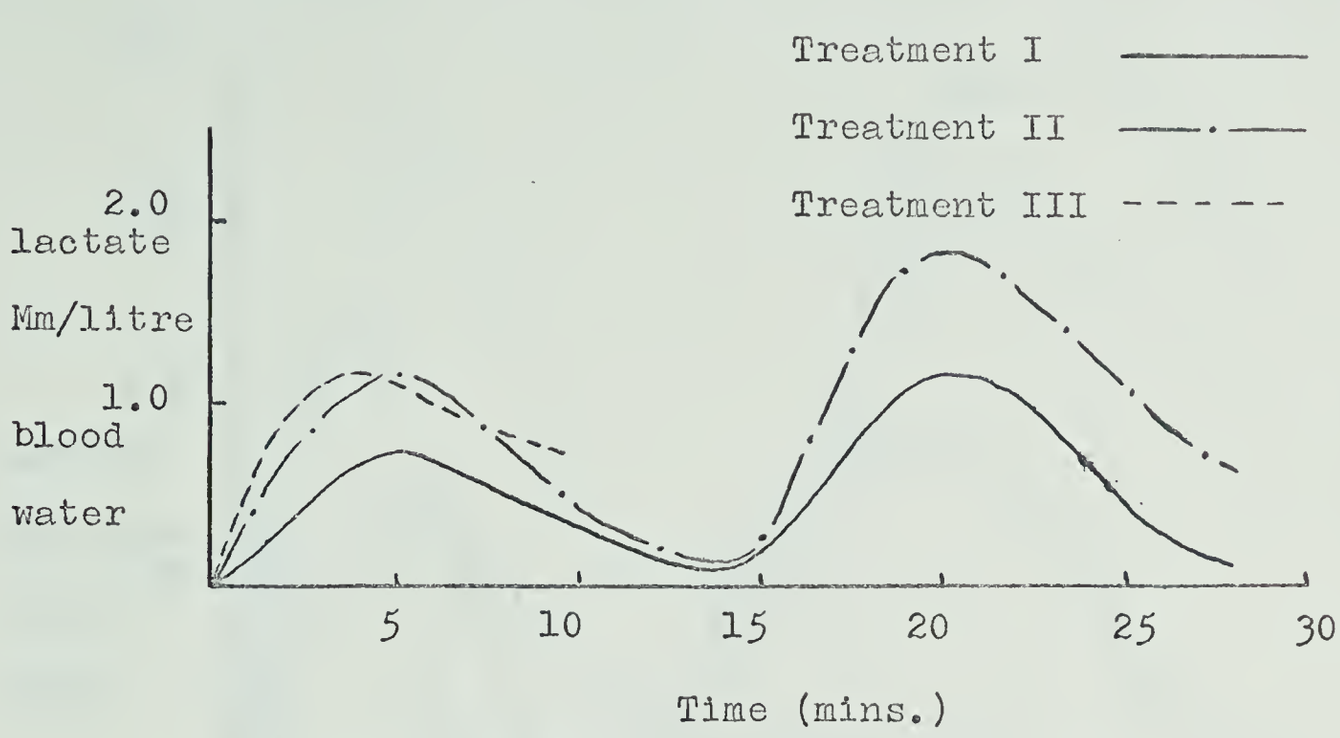


FIGURE III Graphs showing mean values for XL for all treatments



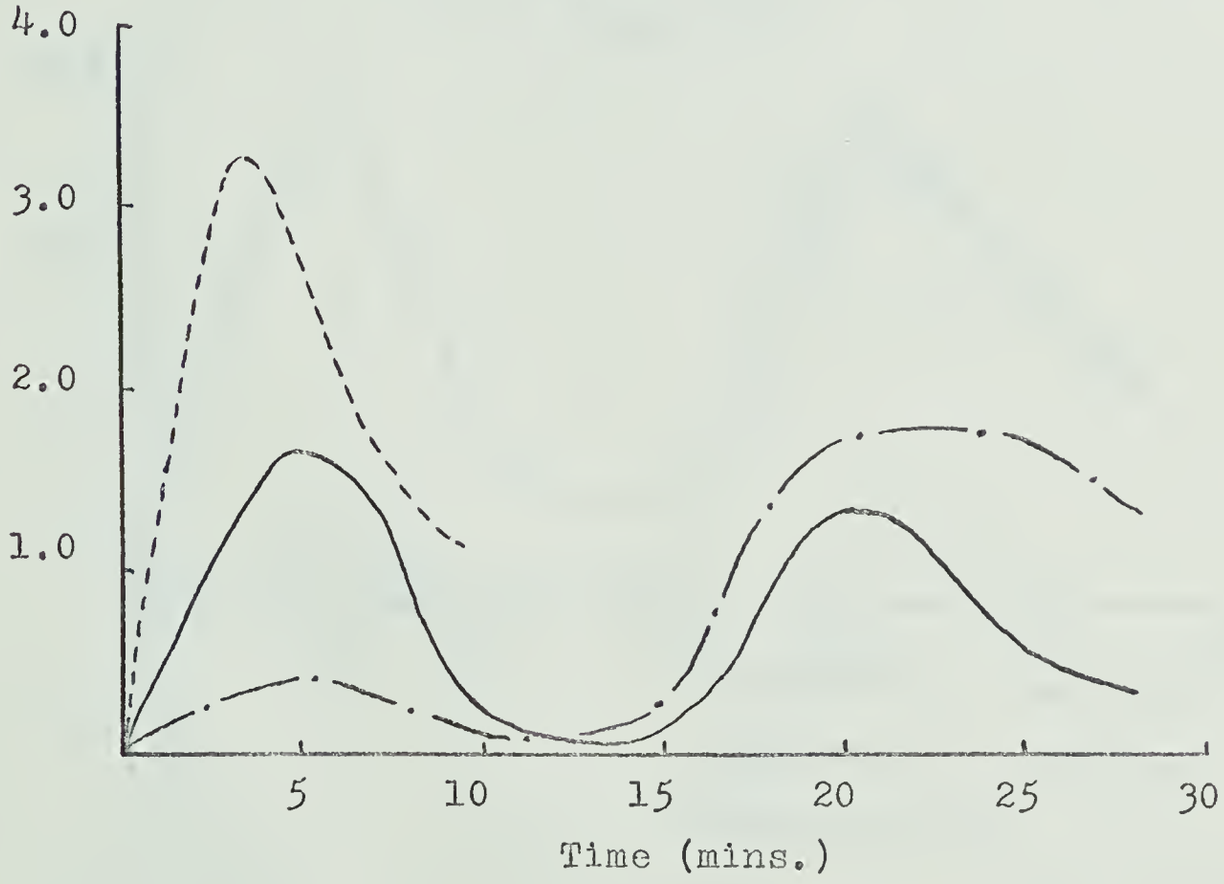


FIGURE IV: 1. Graphs showing XL values for all treatments for subjects 1 (top) and 2 (bottom).

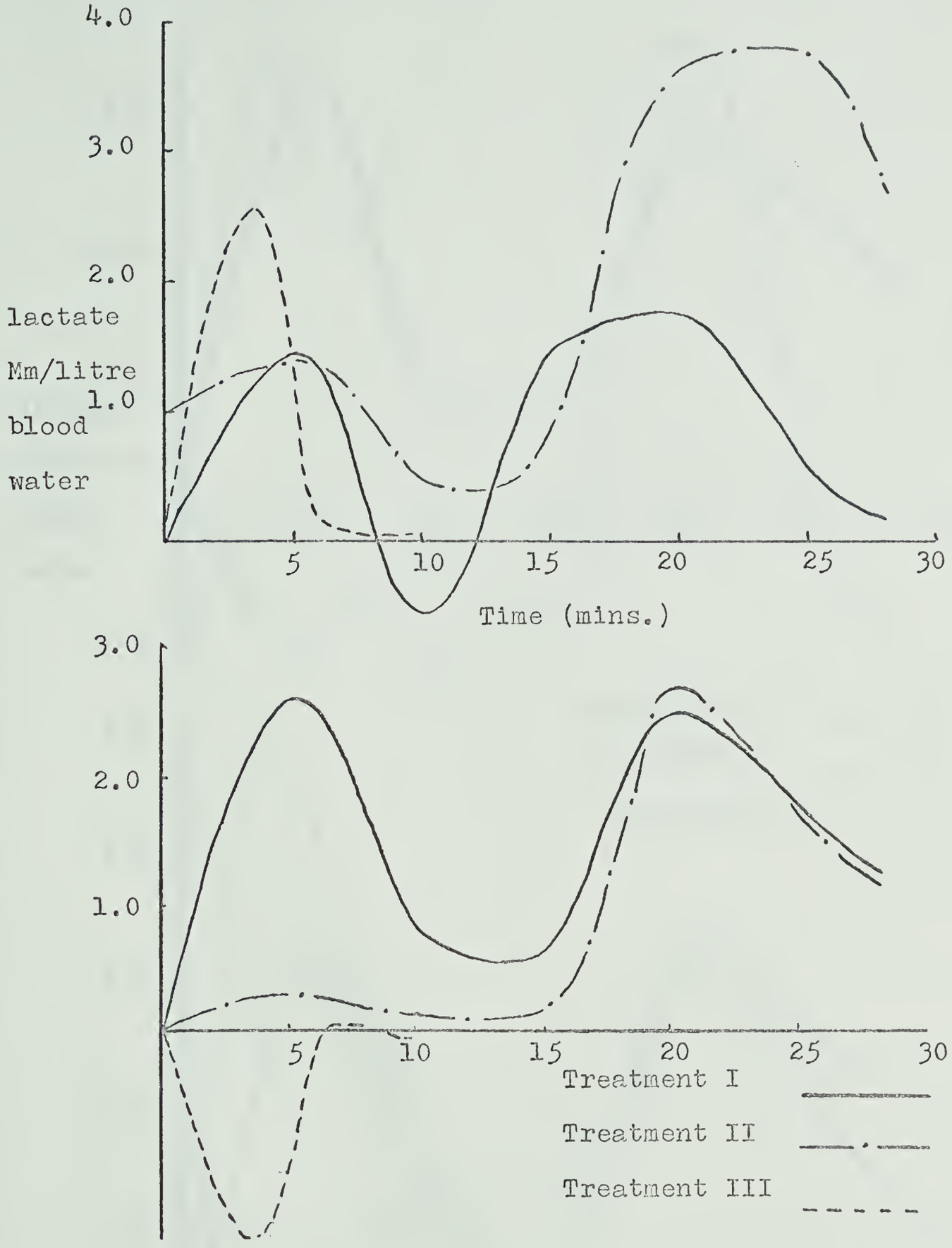


FIGURE IV: 2 Graphs showing XL values for all treatments for subjects 3 (top) and 4 (bottom).

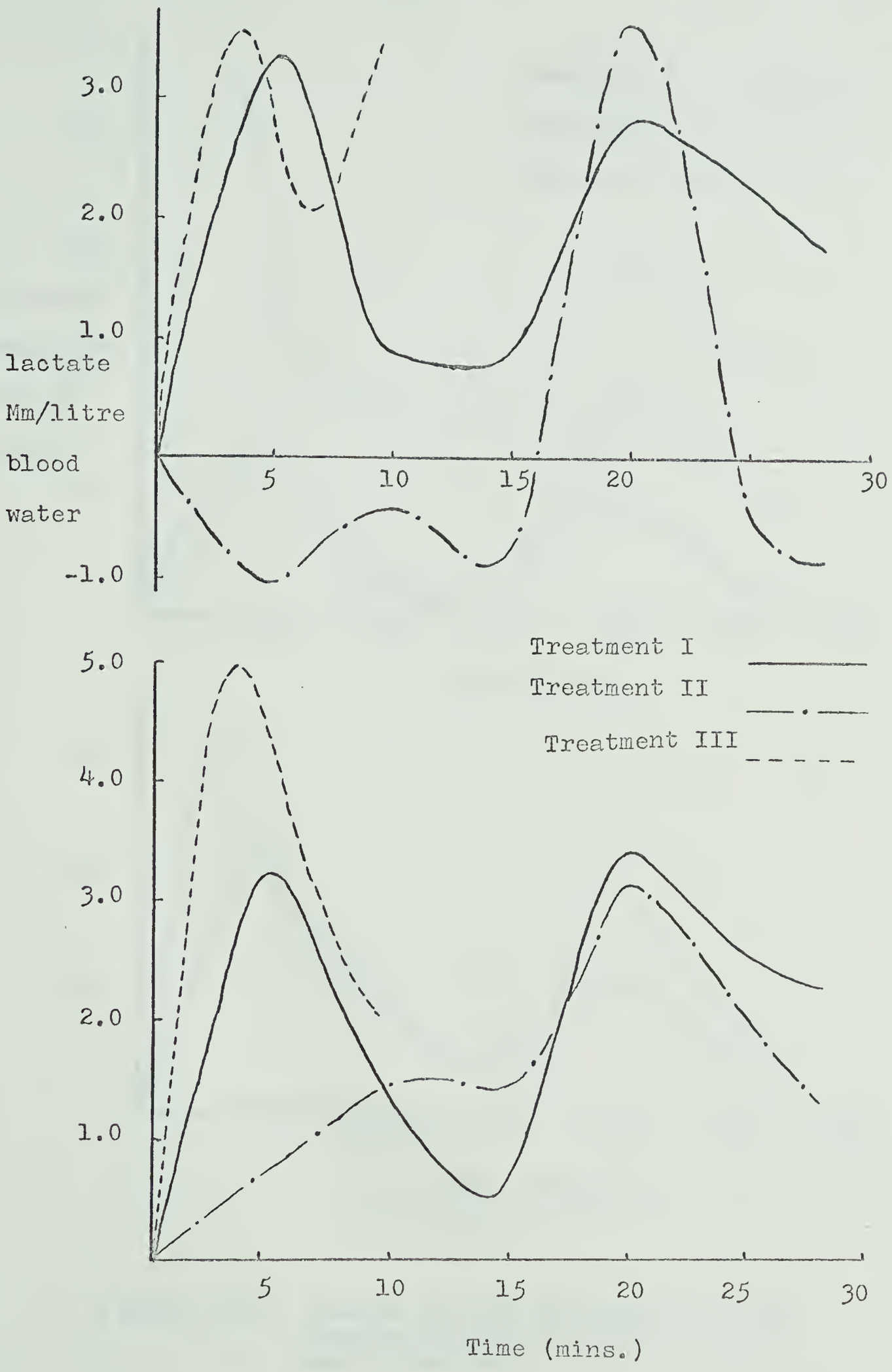


FIGURE IV: 3 Graphs showing XL values for all treatments for subjects 5 (top)

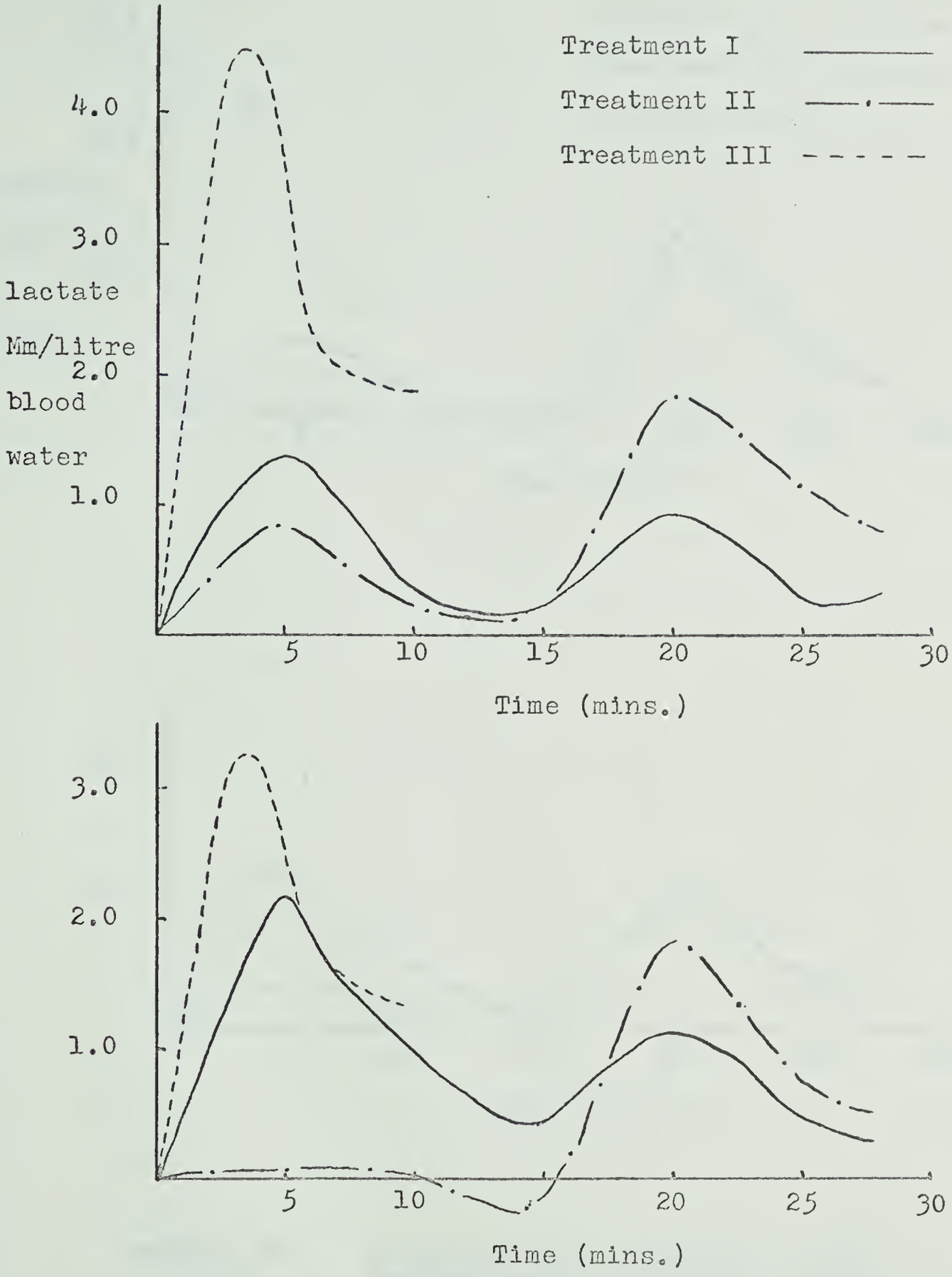
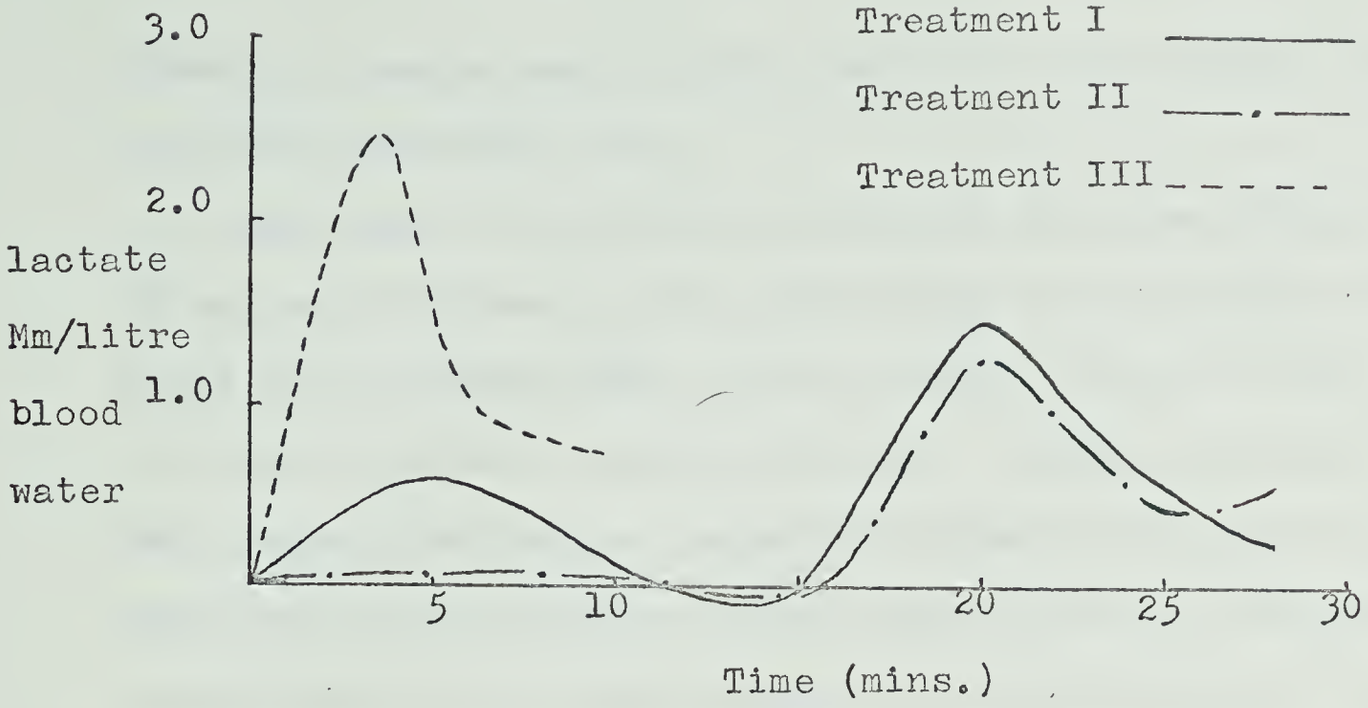


FIGURE IV: 4 Graphs showing XL values for all treatments for subjects 7 (top) and 8 (bottom).



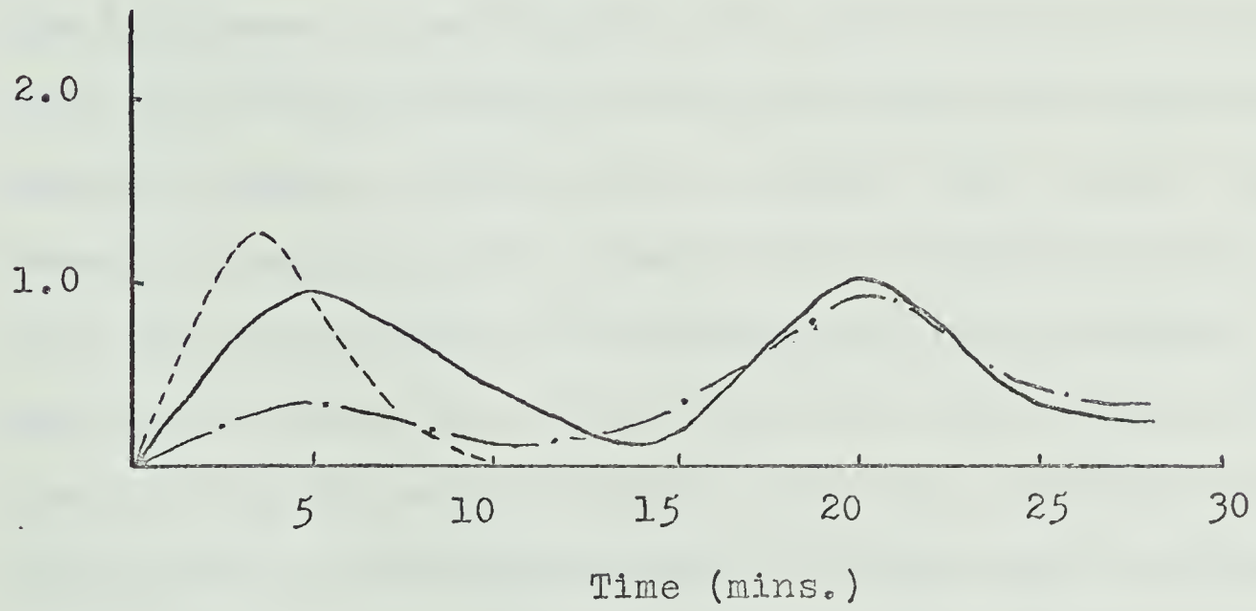


FIGURE IV: 5 Graphs showing XL values for all treatments for subjects 9 (top) and 10 (bottom).

Clearly, a comparison of the graphs for each subject reveals an almost identical form.

Treatment II involved exercise at 55 percent MVO_2 for 90 seconds followed by 30 percent MVO_2 for 15 minutes, followed by 90 percent MVO_2 for 90 seconds. Except in the case of subject number 5 results followed a similar pattern for both lactate and excess lactate to those of Treatment I. Inspection of the graphs clearly shows that the main difference lies in the level reached by ΔL and XL under the first bout of 55 percent MVO_2, the peak value being considerably lower in each case than were reached in the first bout of Treatment I. This is as would be expected from the difference in work levels. In the following 30 percent bout for fifteen minutes, both ΔL and XL levels declined again to resting levels. It is notable that in this case, where the lactate level formed in the initial 90 second bout was not as high as in Treatment I, levels always plateaued between the ten and fourteen minute samples and that in four of the ten subjects the level actually dropped below that of the resting sample, although only by approximately 0.1 mM in each case. This is thus further evidence of lactic acid metabolism occurring during aerobic levels of exercise. In the final 90 second bout of Treatment II levels rose to a point very close to the levels obtained in the ninety percent bout of treatment I, and actually the graph showing the average of this point (figures I and III) shows that it is slightly above the level of Treatment I.

In fact, if one considers that in every case in this final increase the lactate level began at a point somewhat lower than in Treatment I (having returned to resting level), the actual increase in each case is somewhat greater than for the same bout in Treatment I.

Treatment III consisted only of a short, 90 percent MVO_2 exercise bout for one and a half minutes. This treatment was to serve as a check on the 90 percent levels reached in the other treatments. The original hypothesis also suggested that, according to Margaria etal., if an anaerobic phase occurred at the beginning of any exercise period before physiological adjustments took place to serve the new demands placed on the body system, then the amount of lactic acid formed in this period should be greater than if a subject had worked aerobically for some time, and then performed anaerobically in a final burst of exercise.

Inspection of the individual graphs for each subject, whether for ΔL or XL indicates that this hypothesis is correct. In the case of ΔL the peak value of Treatment III exceeds that of Treatment II by 1.44 mM and for XL by .73 mM/litre. The peak values for ΔL (Treatment III) averaged .68 mM/litre higher than those for XL, although as in Treatments I and II the actual shape of the curve in each case appears almost identical.

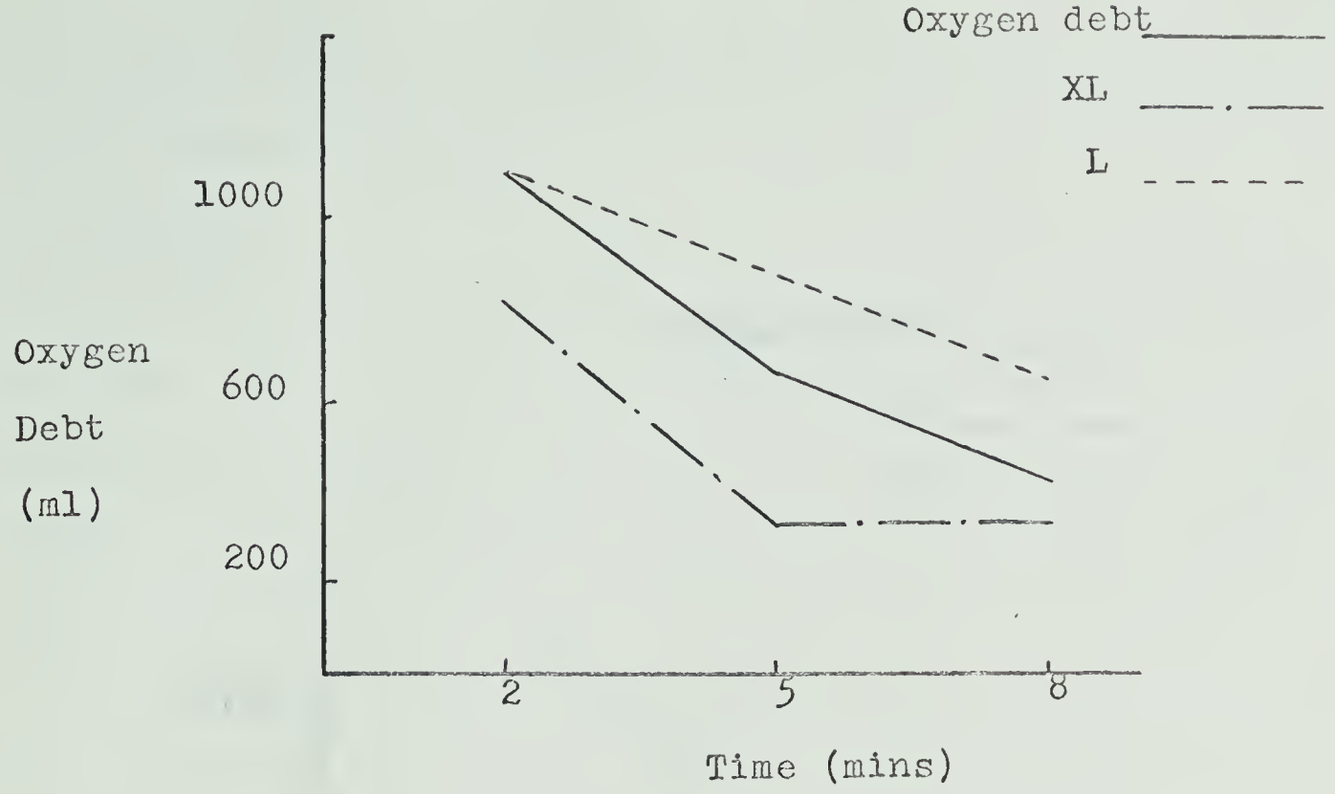
The standard deviations for each point plotted on the average graphs for both Treatment I and II are shown in table I, Appendix 'B'. In each case they are seen to be

quite large. This indicates only that there is quite a large variability between subjects in the amount of lactic acid generated for a particular work level. The graphs indicate, however, that there is little variability between treatments for each subject, i.e. peak levels remain consistently low or high.

Rate of lactic acid metabolism during the aerobic phase compared to that at rest. Since there were only three blood samples collected during the fifteen minute aerobic period it would not be valid to compare the slope of each graph mathematically. However, trends can be obtained by visual inspection.

The decay curve for lactic acid at rest has been shown to be an exponential function with a half time of fifteen minutes (37). Therefore the only Treatment from which a comparison can be made is Treatment I where the peaks for the 90 percent MVO_2 condition approximately coincide. Inspection of each individual graph for ΔL reveals that in every case the slope of the curve in the fifteen minute aerobic period is steeper than in the fifteen minute period immediately following exercise when the subject was sitting resting. This is also born out in the graph showing the average of all conditions. This supports the findings of Edwards and Dill (13) indicating that metabolism of lactic acid occurs at a more rapid rate when a subject is performing light exercise compared to when the subject is resting. The XL curves show similar results.

Lactic acid versus oxygen debt. Huckabee's XL term theoretically calculates only that lactic acid which is due to hypoxia (28). Therefore, if converted to its oxygen equivalent it should correlate very highly with the oxygen debt at any time in exercise or recovery. In this investigation, oxygen was collected in separate bags for the two, five and eight minute time intervals to coincide with the taking of blood samples in the recovery period. It has therefore been possible to compare the oxygen debt values obtained from the gas collection with the oxygen equivalence figures obtained for XL and ΔL, calculated according to the method described in Appendix 'D'. The mean value was calculated for the two, five and eight minute periods for oxygen, XL and ΔL from the table presented in Appendix 'D'. These means were then plotted, and the resulting graphs are shown in figure V. In each case, the ΔL values convert to an oxygen debt which is considerably greater than the values obtained by direct measurement. The XL values as calculated by Huckabee's formula show no coincidence with the oxygen debt values as actually measured, although it is noticeable that the curves show similar shapes. A point of interest is the rise in the measured oxygen debt in the two to five minute collection period of Treatment III, parallelled by a similar rise in the calculated oxygen equivalent of XL. Approximately five percent of the oxygen debt is thought to be associated with the extra circulatory and respiratory effort associated with recovery. In this short bout of intense exercise the rise



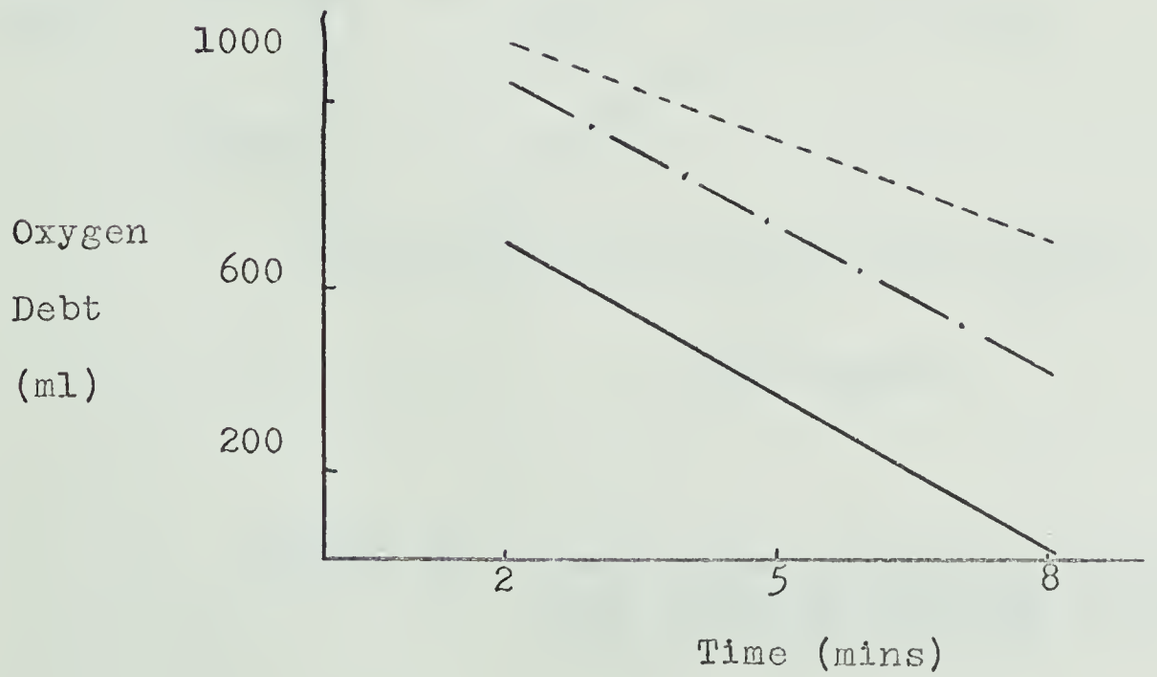


FIGURE V:1 Mean oxygen eqivalent values for L and XL compared to oxygen debt for treatment I (top figure) and treatment II (bottom figure).

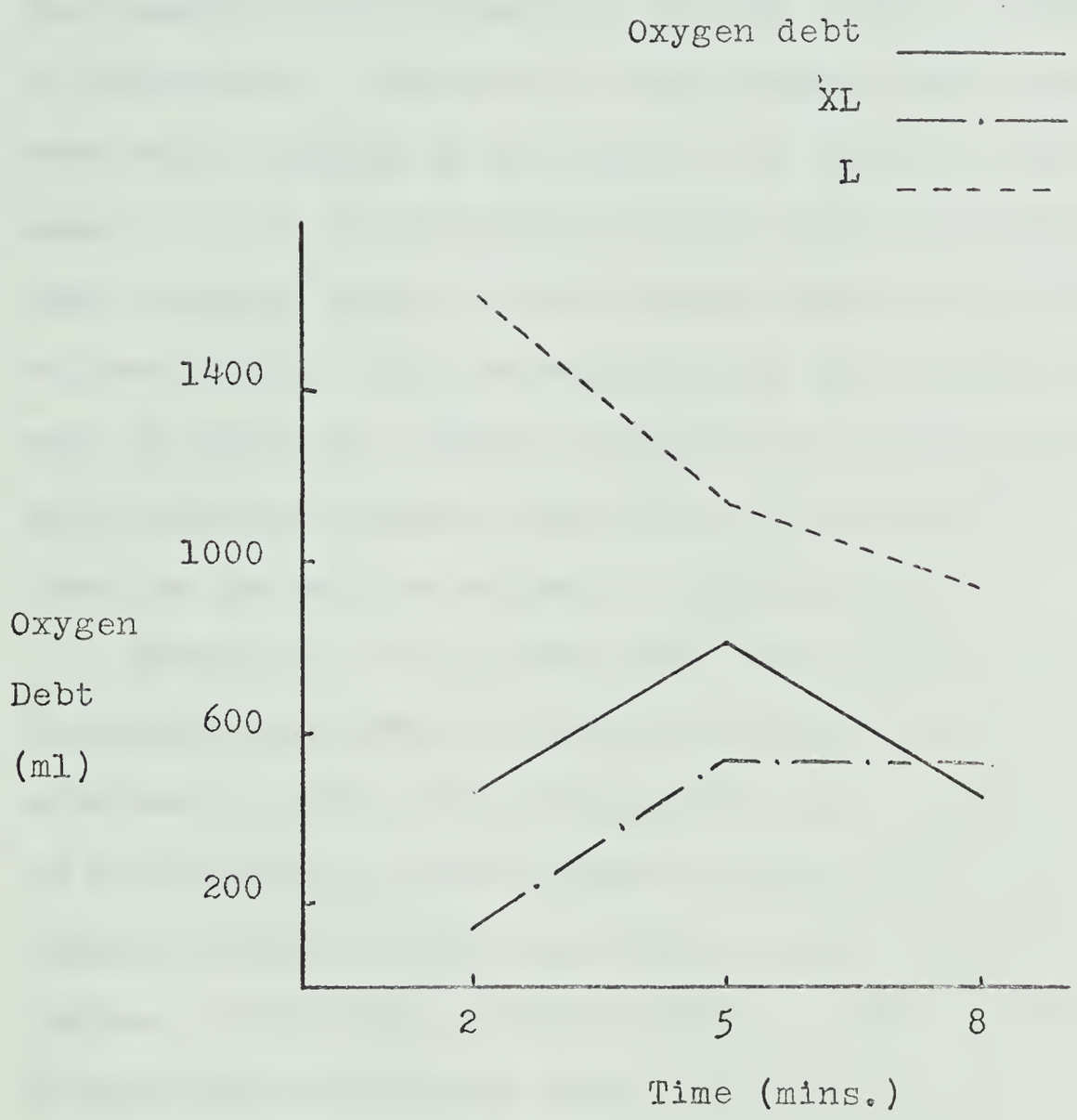


FIGURE V:2 Mean oxygen equivalent values for L and XL compared to oxygen debt for treatment III.

in oxygen debt in the recovery phase mentioned could be a manifestation of this.

Discussion.

The results from treatment I present several aspects worthy of discussion. Firstly, it would appear that lactate values were still falling at the end of the fifteen minute aerobic phase, and it is therefore possible that had this period been slightly longer, these values would have actually reached base level. This is supported by the results from treatment II where the initial increase in lactate was not as great and the fifteen minute time period proved to be sufficient for lactate to return to resting values.

Secondly, it was noted that peak values in the final 90 percent bout did not generally reach the peak values obtained in the initial 90 percent bout. In the Review of Literature, it was mentioned that several recent investigators (13,49,51,52) had noted a small increase in lactate values at the start of any exercise. They attributed this to anaerobic metabolism which occurred while the body adjusted to the increased demands suddenly imposed upon it. The fact that the first 90 percent MVO_2 bout gave higher values than the final one lends support to the above findings since it would be expected that as well as the increase in lactate occurring because of the 90 percent MVO_2 workload, a further increase would take place while the body adjusted as stated above. As a workload of 55 percent MVO_2 falls well within the limits of aerobic work, the initial rise occurring

under the 55 percent condition of treatment II is probably due solely to this fact.

The final 90 percent increase of treatment II rose to a slightly greater peak than for the same bout in treatment I. Why this should be so is difficult to see. Certainly it disproves Huckabees 5 percent of metabolic rate theory convincingly, since if he were correct this final level for treatment II should be considerably lower than that for treatment I. The only explanation which seems plausible is that following the initial 90 percent bout of treatment I the body overcompensated in its reaction to stress and maintained a higher blood oxygen transporting ability for some time following the stress in the event of further heavy exercise. When the subject performed an initial bout at only 55 percent on the other hand overcompensation did not result, or at least not to the same degree. Therefore, when the final 90 percent bout of treatment II commenced a greater time lag occurred before the body adjusted to the greater tissue oxygen demand.

Theoretically, the peak value reached in treatment III (90 percent MVO_2 for 90 seconds) should have coincided with the value obtained for the initial 90 percent period of treatment I, since both are almost maximal, immediate bouts of exercise. The fact that the values for treatment three are higher can be explained if one considers the time interval following exercise that the blood sample was taken.. The samples for treatment three were designed to coincide, for

comparison purposes, with those taken at the conclusion of exercise, and following the 90 percent MVO_2 bouts of treatments I and II, i.e. at two, five and eight minutes. The two and five minute periods were chosen because previous workers had indicated that this was the time that it took for tissue lactate to diffuse into the blood stream and equilibrate in the arterial vessels at some distance from the working muscle.

On the other hand, the samples taken during the exercise period were given an even spread in an attempt to obtain an accurate indication of fluctuations in lactate levels during the aerobic period. Thus, the first sample was taken five minutes after exercise began; i.e. one and a half minutes later than the sample for treatment III. Thus, for the conditions of this experiment it seems that lactate values may have been peaking in arterial blood at some distance from the exercise site at a time somewhere between one and two minutes.

However, it is notable that the peak values for treatment III are somewhat greater than those obtained for either of the other two 90 percent MVO_2 exercise bouts. This is consistent with the statement above, indicating that as well as the 90 percent MVO_2 anaerobic lactate formation, there is a further anaerobic build up at the start of any exercise.

The fact that lactate metabolism is more rapid during light aerobic exercise than at rest was shown not

only for ΔL but also for XL. This finding is contrary to that of Huckabee, but is consistent with that of Wasserman (58) and Edwards (13). Blood lactate levels during muscular work depend upon the rate of formation in the working muscle, the rate of diffusion from the working muscle into the blood and adjacent tissue, rate of transport to sites of utilization and the rate of cellular utilization and excretion of lactate. Rate of transport to sites of utilization is increased by the circulatory increase induced by exercise. The main site of lactate utilization appears to be the heart muscle which uses it as fuel. Some is lost through sweating though this is probably not a factor since it has been shown that lactate concentration in sweat is independent of the plasma level. Seemingly, it is produced through sweat gland metabolism (48). The major site of lactate removal is the liver where gluconeogenesis appears the most likely pathway. That the liver performs this function during exercise, removing as much as 50 percent of lactate formed, has been shown by Rowell etal (51). These findings all lend support to the common practice of many athletes who feel that they recover more quickly between events if they perform light exercise such as jogging in lieu of complete rest.

The results comparing XL, ΔL and oxygen debt would appear to support the findings of other investigators (51,59, 34,30,32,52) which negate any relationship between oxygen debt and lactate levels. They also agree with Wasserman's finding that the total oxygen equivalents of ΔL or XL are

less than the oxygen debt calculated by direct measurement. As Rowell notes (51) at least four factors determine this relationship. They are: the duration of the exercise, blood flow to resting tissue, the capacity of resting tissues to substitute lactate for other substrate and the quantity of lactate produced in relation to the metabolic rate of resting tissues. One would expect that as exercise increased in intensity and duration the quantity of lactate removed by resting tissue would become proportionately smaller. Knuttgen (32) found no correspondence between lactate and oxygen debt where the oxygen debt remained below 1.5 litres. In this study, an inspection of the table in Appendix D reveals that in many cases the oxygen debt did not exceed this value, and that even where it did there is no close agreement between these two values. According to the classical concept of exygen debt in which it cannot be repaid in exercise, one would expect treatment I to have the largest debt, followed by treatment II and then treatment III. However, this is not the case and there appears to be no regular pattern. The large fluctuations obtained for oxygen debt values from the direct measurement may be due to the state of the subject when the resting sample of gas was collected. Generally this was just before cathetarization when oxygen uptake may have been elevated somewhat by anticipation of the procedure to follow.

Finally, the almost identical form of all of the graphs showing rise and fall of ΔL and XL values for each treatment is noteworthy. It indicates that the same processes are occurring in all subjects with respect to lactate metabolism.

However, treatment II for subject 5 does show a departure from the general pattern. It is possible to advance hypothetical reasons for the sharp decline that this subject shows once light exercise began.

Firstly, owing to counterbalancing of trials, treatmentII was the first treatment performed by this subject. An emotional effect may have therefore induced a rise in the initial lactate level. Huckabee has shown that factors other than hypoxia can produce such an elevation in lactate values.

One of these factors was hyperventilation. When the subject reported to the laboratory, he was allowed to rest and then a resting gas sample was collected for three minutes in order to determine basal oxygen consumption. It is possible that some hyperventilation occurred as an effect of this. As the needle was inserted immediately following this, elevated lactate values could have resulted which again would have returned to normal once light exercise began.

Error may have also been introduced during the analysis of lactate and pyruvate. However, this seems unlikely as samples for other subjects were analysed in the same batch and the values obtained gave the characteristic curve when plotted.

CHAPTER V

SUMMARY AND CONCLUSIONS

Summary. This study was designed to determine the effect of varying the exercise intensity from anaerobic to aerobic on blood lactate concentrations. Principally investigated was the hypothesis that a relatively high lactic acid concentration formed by an initial anaerobic exercise bout could be reduced while the subject was still exercising if the exercise was reduced to an aerobic level. Also studied was rate of lactic acid metabolism, both within exercise and at rest following exercise, the difference in information conveyed by "excess lactate" compared to increase in lactate, and the relationship between oxygen debt and lactic acid concentration following exercise.

Subjects each performed three treatment conditions on a bicycle ergometer. Treatment one involved exercising for one and a half minutes at 90 percent MVO_2 followed by fifteen minutes at 30 percent MVO_2, followed by one and a half minutes at 90 percent MVO_2. Treatment II was 55 percent for one and a half minutes, 30 percent for fifteen minutes and 90 percent for one and a half minutes. Treatment III was 90 percent for one and a half minutes. The maximal oxygen uptake for each subject was determined by administering the modified Astrand bicycle ergometer test to each of them. Arterial blood samples were collected throughout exercise and in recovery and analyzed for lactate and pyruvate spec-

trophotometrically. Expired gas was collected for twenty minutes following exercise to calculate oxygen debt. Results were graphed and inspected for main trends.

Conclusions. The following conclusions are supported by the results obtained.

1. A slight increase occurs in blood lactate concentration at the start of any exercise. If the exercise remains aerobic however this subsequently returns to approximately base level.

2. Lactic acid is metabolized in exercise if the exercise is of a low aerobic nature.

3. Lactic acid is metabolized at a faster rate in exercise than it is at rest.

4. The "excess lactate" concept of Huckabee did not add any information additional to that obtainable from increase in lactate alone.

5. There is no relationship between the oxygen debt and increase in lactate following an exercise regime as presented in the three treatments.

Recommendations. Further studies of this nature could be designed to investigate the following:

1. The effect of a longer initial work period to raise the blood lactate level to maximal values, followed by a longer aerobic period to provide sufficient time for lactate levels to return to resting concentrations.

2. The relationship between lactic acid and oxygen debt throughout an exercise period in which the exercise levels

are varied from anaerobic to aerobic in a similar way to the treatments in this study.

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APPENDICES

APPENDIX A

LACTIC AND PYRUVIC ACID DETERMINATION AND COMPUTER PROGRAM

LACTIC ACID-ENZYMATIC METHOD

1. A stock standards are first mad up by dissolving 639.6 mg of DL Lithium Lactate in 100 mls water. This must be kept refrigerated. Before making up working standards the stock solution is diluted 5-25 mls with water (conc. = 60 mg% L.A.). Working standards are then made up as follows.

Mls diluted working stds.	0	0.5	1.0	1.5	2.0	2.5	3.0
mls H_2O	3	2.5	2.0	1.5	1.0	0.5	0.0
mls. TCA 10%	5	5	5	5	5	5	5
Lactate (mgm/0.1ml SNF)	0	3.75	7.5	11.25	15.0	18.75	22.5

2. A glycine hydrazine buffer is made by dissolving 37.5 gm glycine in approximately 800 mls of distilled water, adding 13.5 mls. hydrazine (S.G. 1.011, 95%) mixing well and adjusting pH to 9.0 with 2.5N NaOH. It is then diluted to 1.000 mls. with water, and kept for two weeks in the refrigerator.

3. Lactic acid Dehydrogenase is diluted on the day of use 0.4 ml. LDH conc. made up to 160 mls with glycine hydrazine buffer.

4. DPN (cozymase) is diluted 30 mg/ml H_2O.

5. Test tubes are set up in order with two empty tubes per sample tube plus seven tubes for the standards. Blood samples had previously been centrifuged. Into each of the empty tubes is measured 4 mls of Glycine Hydrazine Buffer plus LDH. Using a .2 ml. pipette, .2 ml of SNF (supernatant fluid) of each speciman is added to the Glycine Hydrazine Buffer and

mixed well.

6. At zero time 0.4 mls D.P.N. are added to the set of duplicate blanks, and thereafter consecutively to the remaining tubes. The tubes are shaken after the addition of D.P.N. and allowed to remain at 25-26 degrees Centigrade for exactly one hour.

7. Two optical density readings are then taken at 335 mu against distilled water using the UNICAM spectrophotometer. The average of the two readings is read as the final value for lactic acid.

Calculations:

1. From the readings for the 7 standards a standard curve is plotted. This gives the lactate result in Mgm/0.1 mls SNF

Then:

$$\text{Lactic Acid (mg\%)} = \frac{(\text{Result in mgm/0.1 ml SNF}) \times (\text{Vol TCA} \div \text{Vol. blood}) \times 100 \text{ ml}}{\text{Volume of blood}}$$

$$\text{Lactic Acid (mg\%)} = \frac{(\text{T in Mgm/0.1 ml SNF}) \times (5 \text{ ml} \div \text{V mls}) \times 100 \text{ ml}}{\text{V mls}}$$

$$\text{Lactic Acid (mg\%)} = \frac{T \times (5 \div V)}{V}$$

To convert units to millimoles/litre:

G.M.W. of lactic acid = 90.08

Then 1 mole of Lactic Acid = 90.08 gms

1 gm = 1/90.08 moles/ millilitre

Therefore 1 mgm = 1/90.08 millimoles

Then, as results obtained above were in mgm/100 ml blood to convert x mgm/100 ml blood to millimoles/litre blood

=(x) x 10/90.08 millimoles/litre blood

To convert to millimoles/litre of blood water:

2 ml blood contain x ml water

Therefore, 1 ml blood contains x/2 ml water

= ((x) x 10)/(90.08 x blood water(ml) x .05) millimoles/ litre blood wtr.

PYRUVIC ACID-CHEMICAL METHOD

Reagents.

1. 10% TCA (refrigerated).

2. 0.1% D.N.P.H. made by grinding 100 mg 2:4 dinitrophenylhydrazine repeatedly in a mortar with volumes of about 5 ml 2NHCL. The supernatuant is decanted into a 100 ml measuring cylinder and this process continued until the 100 ml mark is reached.

3. Toluene.

4. 10% Na_2CO_3.

5. 1.5N NaOH.

6. The stock standard is made by dissolving 375 mg of sodium pyruvate in 100 ml H_2O to give the equivalent to 300 mg% pyruvic acid.

7. From the stock standard, working standards are made as follows.

   a. Dilute stock 1:100 with water prior to use, to give 3 mg% pyruvic acid.

   b. Standards are then made up as follows:

mls working standard	0	0.5	1.0	1.5	2.0	2.5	3.0
mls H_2O	3.0	2.5	2.0	1.5	1.0	0.5	0.0
mls TCA 10%	5	5	5	5	5	5	5
Pyruvate (mgm/2ml SNF)	0	3.75	7.5	11.25	15.0	18.75	22.5

Procedure.

1. Into duplicate stoppered centrifuge tubes pipette 2mls SNF of sample, 2 mls TCA diluted standards, and 2 mls blank (0mg%

of sample, 2 mls TCA diluted standards, and 2 mls blank (0mg% P.A. std.).

2. Allow tubes to equilibrate at room temperature.

3. Add 1 ml 0.1% D.P.N.H. reagent at 30 second intervals. Allow each tube to stand for exactly 5 minutes.

4. Add 2 mls Toluene. Stopper tube immediately and extract hydrazone by inverting 10 times.

5. With a Pasteur pipette, aspirate the lower layer and discard.

6. Add 2.5 mls 10% Na_2CO_3, stopper and shake well for 1 min..

7. Transfer 2 mls of lower layer (Na_2CO_3) to a small test tube in which is 2 mls 1.5N NaOH. Mix to develop colour.

8. Read O.D. at 520 mu on UNICAM model 'B' spectrophotometer.

Calculations.

1. From the readings for the 7 standards a standard curve is plotted. This will give pyruvate result in gm/2 ml SNF.

Then pyruvic acid (mg%) =

$$\frac{(\text{result in gm/2ml gm/2ml SNF}) \times (\text{Vol TCA} + \text{Vol blood}) \times 100\text{ml}}{\text{Vol blood}}$$

=

$$\frac{(\text{T in gm/2ml SNF})(5\text{mls} + \text{V mls}) \times 100\text{ ml}}{\text{V mls}}$$

$$= \frac{\text{T} \times (5 + \text{V})}{20\ \text{V}} \quad \text{mg\%}$$

This value is then converted to millimoles/ litre as was done for lactic acid.

G.M.W. for pyruvic acid = 88.06.

```
LEVEL 1, MOD 4                MAIN                    DATE = 70207          15/24/36

      DIMENSION NA(186),NO(186),TBW(10),FLAC(186),FPYR(186),XLAC(186),
     1DLAC(186),O2E(186),DO2E(186),BLWTR(10),AGE(10),BWT(10),WT(186),DEN
     7(186),ALAC(186),SLL(186),PYR(186),SLP(186)
   17 FORMAT(1H,5X,4HNAME,4X,11HBLOOD WATER,4X,16HTOTAL BODY WATER,4X,6H
     8WEIGHT,4X,3HAGE)
      WRITE(6,17)
      DO 11 L=1,10
      READ(5,3)NX,BWT(L),AGE(L),BLWTR(L)
    3 FORMAT(I2,F5.2,F4.2,F5.3)
      IF(NX.EQ.77)GO TO 4
      BWT(L)=BWT(L)*.4545
      TBW(L)=(79.45*BWT(L)/100)-((0.24*(BWT(L)**2))/100)-(0.15*AGE(L)*BW
     4T(L)/100)
   16 FORMAT(6X,I2,8X,F6.3,10X,F6.3,12X,F3.0,6X,F3.0)
      WRITE(6,16)NX,BLWTR(L),TBW(L),BWT(L),AGE(L)
   11 CONTINUE
    4 DO 6 I=1,186,1
      READ(5,2)NA(I),NO(I),WT(I),DEN(I),ALAC(I),SLL(I),PYR(I),SLP(I)
    2 FORMAT(I2,I2,F6.5,F6.5,F4.2,F4.3,F4.2,F4.4)
      IF(NA(I).EQ.77)GO TO 14
      VOL=WT(I)/DEN(I)
      TLAC=ALAC(I)/SLL(I)
      RLAC=TLAC*(5+VOL)/VOL
      K=NA(I)
      FLAC(I)=RLAC*10.0/(90.08*BLWTR(K)*0.5)
      IF(NO(I).EQ.11)OLAC=FLAC(I)
      IF(NO(I).EQ.12)OLAC=FLAC(I)
      IF(NO(I).EQ.13)OLAC=FLAC(I)
      TPYR=PYR(I)/SLP(I)
      RPYR=TPYR*(5+VOL)/(VOL*20)
      FPYR(I)=RPYR*10.0/(88.06*BLWTR(K)*0.5)
      IF(NO(I).EQ.11)OPYR=FPYR(I)
      IF(NO(I).EQ.12)OPYR=FPYR(I)
      IF(NO(I).EQ.13)OPYR=FPYR(I)
      DLAC(I)=FLAC(I)-OLAC
      XLAC(I)=DLAC(I)-(FPYR(I)-OPYR)*(OLAC/OPYR)
      IF(NO(I).EQ.23)GO TO 8
      IF(NO(I).EQ.33)GO TO 8
      IF(NO(I).EQ.43)GO TO 8
      IF(NO(I).EQ.51)GO TO 8
      IF(NO(I).EQ.61)GO TO 8
      IF(NO(I).EQ.71)GO TO 8
      IF(NO(I).EQ.52)GO TO 8
      IF(NO(I).EQ.62)GO TO 8
      IF(NO(I).EQ.72)GO TO 8
      GO TO 6
```

```
 8 O2E(I)=TBW(K)*11.2*XLAC(I)
   DO2E(I)=TBW(K)*11.2*DLAC(I)
 6 CONTINUE
14 WRITE(6,15)
15 FORMAT(1H,13X,2HSB,1X,2HNO,3X,2HWT,5X,1HD,4X,1HL,4X,2HSL,3X,1HP,4X
  9,1HS,4X,2HLA,3X,2HIL,3X,2HXL,3X,2HPY,5X,3HO2X,4X,3HO2I)
   DO 19 M=1,186
   WRITE(6,18)NA(M),NO(M),WT(M),DEN(M),ALAC(M),SLL(M),PYR(M),SLP(M),F
  1LAC(M),DLAC(M),XLAC(M),FPYR(M),O2E(M),DO2E(M)
18 FORMAT(13X,I2,1X,I2,1X,F6.4,1X,F6.4,1X,F4.1,1X,F4.2,1X,F4.1,1X,F4.
  22,1X,F4.1,1X,F4.1,1X,F5.2,1X,F4.1,1X,F6.1,1X,F6.1)
19 CONTINUE
20 STOP
   END
```

APPENDIX B

LACTATE/PYRUVATE DATA

KEY TO DATA COLUMNS

1. SB_____Subject numbers
2. NO_____Code for treatment and sample number. e.g. 11 is the first sample of treatment I, 23 is the second sample of treatment III.
3. WT_____Weight of blood sample taken
4. D_____Density of subject's blood
5. L_____Spectrophotometric reading of lactate
6. SL_____Slope obtained by plotting lactate standards read on spectrophotometer
7. P_____Spectrophotometric reading of pyruvate
8. S_____Slope obtained by plotting lactate standards read on spectrophotometer.
9. LA_____Lactate Mm/litre of blood water, calculated as described in Appendix 'A'
10. IL_____Increase in lactate Mm/litre of blood water (ΔL)
11. XL_____Excess lactate in Mm/ litre of blood water, calculated according to Huckabees formulae.
12. PY_____Pyruvate Mm/litre of blood water, claculated as described in Appendix 'A'
13. O_2X_____Oxygen equivalent of excess lactate, calculated as described in Appendix 'D'
14. O_2I_____Oxygen equivalent of increased lactate, calculated as described in Appendix 'D'

NAME	BLOOD WATER	TOTAL BODY WATER	WEIGHT	AGE
1	1.587	46.522	84.	25.
2	1.595	38.859	65.	25.
3	1.577	41.904	71.	24.
4	1.597	44.975	79.	24.
5	1.553	40.946	69.	24.
6	1.605	46.110	83.	27.
7	1.598	42.726	74.	25.
8	1.606	43.923	76.	24.
9	1.597	38.859	65.	25.
10	1.624	42.685	73.	22.

SB	NO	WT	D	L	SL	P	S	LA	IL	XL	PY	O2X	O2I
1	11	3.5225	1.0716	7.0	2.41	12.1	0.54	1.0	0.0	0.0	0.4	-0.0	0.0
1	21	2.4636	1.0716	13.9	2.41	17.1	0.54	2.6	1.5	0.74	0.7	0.0	******
1	31	2.2580	1.0716	10.8	2.41	15.8	0.54	2.1	1.1	0.32	0.7	0.0	******
1	41	3.4376	1.0716	8.3	2.41	13.3	0.54	1.2	0.2	0.09	0.5	0.0	******
1	51	4.5878	1.0716	20.7	2.41	20.0	0.54	2.6	1.6	1.15	0.6	598.7	823.5
1	61	3.5978	1.0716	14.5	2.41	20.0	0.54	2.1	1.1	0.42	0.7	220.6	557.9
1	71	3.7756	1.0716	13.0	2.41	21.1	0.54	1.8	0.8	0.11	0.7	58.1	417.4

2	11	4.8192	1.0773	7.2	2.41	10.8	0.54	0.9	0.0	0.0	0.3	0.0	-0.0
2	21	3.5498	1.0773	29.0	2.41	26.3	0.54	4.2	3.3	1.67	0.9	0.0	******
2	31	3.3390	1.0773	15.0	2.41	20.2	0.54	2.3	1.4	0.23	0.7	0.0	-0.0
2	41	4.5213	1.0773	11.9	2.41	17.3	0.54	1.5	0.6	0.05	0.5	0.0	******
2	51	4.5585	1.0773	27.0	2.41	24.8	0.54	3.4	2.5	1.32	0.7	574.0	1097.5
2	61	4.0343	1.0773	20.2	2.41	24.3	0.54	2.7	1.8	0.54	0.7	234.8	802.5
2	71	3.7383	1.0773	15.1	2.41	19.1	0.54	2.1	1.2	0.33	0.6	145.2	543.2
3	11	4.0537	1.0724	6.2	2.41	10.3	0.54	0.8	0.0	0.0	0.3	0.0	******
3	21	4.0629	1.0724	25.7	2.41	25.3	0.54	3.5	2.6	1.42	0.8	0.0	******
3	31	3.8373	1.0724	13.2	2.41	28.2	0.54	1.8	1.0	-0.53	0.9	0.0	******
3	41	3.1475	1.0724	18.2	2.41	15.0	0.54	2.9	2.0	1.45	0.5	0.0	******
3	51	2.5895	1.0724	21.0	2.41	24.0	0.54	3.8	2.9	1.18	1.0	551.7	1373.1
3	61	2.6650	1.0724	19.8	2.41	28.0	0.54	3.5	2.6	0.52	1.1	243.3	1240.3
3	71	0.6976	1.0724	5.3	2.41	8.2	0.54	2.7	1.8	0.18	0.9	86.7	867.4
4	13	4.1257	1.0760	8.5	2.20	19.0	0.57	1.2	0.0	0.0	0.5	-0.0	0.0
4	23	4.1289	1.0760	12.6	2.20	52.8	0.57	1.8	0.6	-1.60	1.5	-807.9	300.3
4	33	5.0066	1.0760	9.5	2.20	20.8	0.57	1.2	0.0	0.03	0.5	12.9	4.0
4	43	4.3458	1.0760	7.6	2.20	17.5	0.57	1.1	-0.2	-0.03	0.5	-16.3	-82.0
5	12	2.6775	1.0834	17.7	2.20	23.8	0.57	3.5	0.0	0.0	0.9	******	-0.0
5	22	3.1824	1.0834	17.8	2.20	31.9	0.57	3.1	-0.4	-1.04	1.1	0.0	******
5	32	3.7251	1.0834	18.1	2.20	27.8	0.57	2.9	-0.6	-0.41	0.9	******	******
5	42	3.1764	1.0834	2.2	2.20	9.8	0.57	0.4	-3.1	-0.89	0.3	******	******
5	52	4.6340	1.0834	43.7	2.20	24.4	0.57	6.2	2.7	3.60	0.7	1651.7	1230.0
5	62	4.0436	1.0834	28.9	2.20	42.9	0.57	4.4	0.9	-0.46	1.3	-209.5	420.4
5	72	5.3352	1.0834	26.6	2.20	44.5	0.57	3.5	0.0	-0.85	1.2	-390.1	2.9
6	13	3.0993	1.0727	5.0	2.20	13.7	0.57	0.9	0.0	0.0	0.5	******	******
6	23	4.6253	1.0727	49.0	2.20	34.1	0.57	6.7	5.8	4.96	0.9	2563.5	2992.9
6	33	4.8464	1.0727	40.4	2.20	43.1	0.57	5.4	4.5	3.27	1.1	1687.7	2320.4
6	43	5.2919	1.0727	33.0	2.20	46.1	0.57	4.2	3.3	2.05	1.2	1057.6	1714.3
7	12	3.1845	1.0734	4.0	2.20	13.6	0.57	0.7	0.0	0.0	0.5	******	******
7	22	4.6366	1.0734	13.3	2.20	23.5	0.57	1.8	1.1	0.87	0.6	******	******
7	32	3.9271	1.0734	7.2	2.20	19.2	0.57	1.1	0.4	0.23	0.6	0.0	******
7	42	4.1574	1.0734	5.9	2.20	16.8	0.57	0.9	0.2	0.14	0.5	0.0	0.1
7	52	4.1672	1.0734	20.3	2.20	24.8	0.57	2.9	2.3	1.88	0.7	899.3	1079.0
7	62	5.1969	1.0734	20.1	2.20	37.9	0.57	2.6	1.9	1.15	1.0	550.0	910.2
7	72	5.3370	1.0734	16.7	2.20	35.0	0.57	2.1	1.4	0.81	0.9	388.3	687.6

8	13	3.0134	1.0725	5.3	2.20	16.1	0.57	0.9	0.0	0.0	0.6	-0.1	******
8	23	5.6651	1.0725	37.6	2.20	33.3	0.57	4.6	3.7	3.26	0.8	1602.9	1807.2
8	33	4.2326	1.0725	25.0	2.20	39.9	0.57	3.6	2.6	1.69	1.1	831.5	1296.6
8	43	4.9543	1.0725	22.7	2.20	37.1	0.57	3.0	2.0	1.37	1.0	675.1	1005.9
1	12	3.8987	1.0716	5.2	2.20	15.5	0.57	0.8	0.0	0.0	0.5	4.3	0.0
1	22	4.8824	1.0716	13.9	2.20	15.1	0.57	1.9	1.1	1.18	0.4	0.3	-0.0
1	32	3.6683	1.0716	8.8	2.20	18.1	0.57	1.4	0.6	0.43	0.6	-0.0	11.0
1	42	3.4200	1.0716	6.0	2.20	16.0	0.57	1.0	0.2	0.10	0.5	******	0.0
1	52	4.8758	1.0716	21.8	2.20	24.5	0.57	2.9	2.1	1.81	0.7	944.5	1107.0
1	62	5.1667	1.0716	17.4	2.20	27.8	0.57	2.3	1.5	1.05	0.7	544.9	765.3
1	72	5.0116	1.0716	13.0	2.20	24.8	0.57	1.7	0.9	0.62	0.6	320.9	482.2
2	12	3.2277	1.0773	3.0	2.20	12.1	0.57	0.5	0.0	0.0	0.4	1.0	******
2	22	2.9584	1.0773	6.1	2.20	15.4	0.57	1.1	0.6	0.41	0.5	0.1	******
2	32	3.3938	1.0773	4.0	2.20	13.3	0.57	0.7	0.1	0.11	0.4	******	******
2	42	3.7561	1.0773	4.5	2.20	14.3	0.57	0.7	0.2	0.15	0.4	-0.0	-0.0
2	52	1.5465	1.0773	9.7	2.20	13.9	0.57	2.8	2.2	1.77	0.8	772.0	977.0
2	62	3.2935	1.0773	16.5	2.20	24.8	0.57	2.8	2.2	1.73	0.8	751.3	977.0
2	72	2.9324	1.0773	12.6	2.20	21.2	0.57	2.3	1.8	1.32	0.8	573.7	763.9
6	11	2.6619	1.0727	4.1	2.20	13.5	0.57	0.8	0.0	0.0	0.5	15.9	******
6	21	3.3667	1.0727	28.5	2.20	28.6	0.57	4.6	3.9	3.23	0.9	0.0	******
6	31	4.1509	1.0727	17.5	2.20	28.0	0.57	2.5	1.7	1.30	0.8	0.0	******
6	41	3.9033	1.0727	10.1	2.20	21.2	0.57	1.5	0.7	0.55	0.6	-0.2	******
6	51	3.6024	1.0727	30.8	2.20	29.7	0.57	4.8	4.0	3.41	0.9	1760.3	2087.9
6	61	3.5936	1.0727	27.9	2.20	38.1	0.57	4.4	3.6	2.56	1.2	1321.6	1856.8
6	71	3.5138	1.0727	23.6	2.20	30.0	0.57	3.7	3.0	2.30	0.9	1188.7	1534.7
7	13	4.9113	1.0734	5.9	2.20	18.4	0.57	0.8	0.0	0.0	0.5	******	6.0
7	23	3.9492	1.0734	38.9	2.20	27.0	0.57	5.8	5.0	4.51	0.8	2156.1	2400.1
7	33	3.8773	1.0734	27.9	2.20	41.6	0.57	4.2	3.4	2.19	1.2	1049.2	1637.2
7	43	4.3614	1.0734	27.2	2.20	42.7	0.57	3.8	3.1	1.90	1.2	910.6	1460.4
5	13	3.2466	1.0834	4.8	2.20	15.0	0.53	0.8	0.0	0.0	0.6	******	******
5	23	3.8546	1.0834	32.6	2.20	31.3	0.53	5.1	4.3	3.53	1.0	1618.9	1955.1
5	33	3.6888	1.0834	24.0	2.20	35.1	0.53	3.8	3.0	2.05	1.2	939.3	1383.8
5	43	4.3567	1.0834	23.5	2.20	0.0	0.53	3.4	2.6	3.43	0.0	1571.1	1189.4
3	12	2.9279	1.0724	2.5	2.20	9.1	0.53	0.5	0.0	0.0	0.4	******	11.7
3	22	3.9681	1.0724	14.8	2.20	20.0	0.53	2.2	1.8	1.40	0.6	0.0	******
3	32	2.9649	1.0724	6.8	2.20	15.2	0.53	1.2	0.8	0.47	0.6	******	******

3	42	3.2938	1.0724	6.9	2.20	13.9	0.53	1.2	0.7	0.52	0.5	******	-0.0
3	52	3.0114	1.0724	26.4	2.20	22.0	0.53	4.7	4.2	3.62	0.8	1700.0	1992.1
3	62	3.7910	1.0724	33.5	2.20	33.0	0.53	5.2	4.7	3.78	1.1	1771.8	2216.7
3	72	3.3485	1.0724	24.4	2.20	30.2	0.53	4.1	3.6	2.68	1.1	1258.1	1693.7
8	11	2.2424	1.0725	4.8	2.20	11.2	0.53	1.0	0.0	0.0	0.5	******	0.0
8	21	4.0008	1.0725	26.9	2.20	28.0	0.53	4.0	2.9	2.19	0.9	******	-0.0
8	31	4.3339	1.0725	15.9	2.20	20.5	0.53	2.2	1.2	1.00	0.6	2823.3	0.0
8	41	4.9007	1.0725	11.8	2.20	19.9	0.53	1.6	0.5	0.43	0.6	11.7	******
8	51	3.4913	1.0725	15.9	2.20	20.0	0.53	2.5	1.5	1.17	0.7	574.5	743.3
8	61	3.9053	1.0725	13.8	2.20	24.5	0.53	2.1	1.0	0.49	0.8	242.1	509.2
8	71	3.0266	1.0725	10.0	2.20	19.2	0.53	1.7	0.7	0.31	0.7	151.8	353.6
9	13	2.6360	1.0746	6.0	2.38	12.7	0.53	1.1	0.0	0.0	0.5	******	0.0
9	23	3.0558	1.0746	24.8	2.38	20.1	0.53	4.0	2.9	2.47	0.7	1073.2	1275.6
9	33	3.0303	1.0746	19.5	2.38	28.8	0.53	3.2	2.1	0.95	1.1	415.5	911.3
9	43	3.5152	1.0746	17.8	2.38	27.0	0.53	2.6	1.6	0.75	0.9	324.2	680.8
4	11	3.7353	1.0760	4.8	2.38	12.0	0.53	0.7	0.0	0.0	0.4	-0.2	0.0
4	21	3.4499	1.0760	27.1	2.38	24.6	0.53	4.1	3.4	2.58	0.8	******	******
4	31	3.9177	1.0760	15.1	2.38	23.9	0.53	2.1	1.4	0.77	0.8	******	******
4	41	4.0340	1.0760	11.0	2.38	17.8	0.53	1.5	0.8	0.53	0.6	******	******
4	51	2.8613	1.0760	23.4	2.38	22.0	0.53	3.9	3.3	2.46	0.9	1237.3	1638.5
4	61	2.6062	1.0760	18.9	2.38	23.2	0.53	3.4	2.7	1.72	1.0	867.4	1359.5
4	71	4.2279	1.0760	20.9	2.38	28.9	0.53	2.8	2.1	1.24	0.9	624.5	1052.8
2	13	3.4104	1.0773	7.6	2.38	15.7	0.53	1.1	0.0	0.0	0.5	0.1	0.0
2	23	2.7813	1.0773	29.7	2.38	22.0	0.53	5.1	4.0	3.27	0.9	1424.1	1721.2
2	33	3.4503	1.0773	27.6	2.38	31.9	0.53	4.1	3.0	1.82	1.1	792.6	1300.4
2	43	3.2311	1.0773	21.5	2.38	29.1	0.53	3.4	2.2	1.16	1.0	503.3	960.6
1	13	7.1215	1.0716	15.8	2.38	17.0	0.53	1.6	0.0	0.0	0.4	1.1	-0.0
1	23	3.5222	1.0716	25.5	2.38	19.0	0.53	3.8	2.2	1.16	0.6	605.5	1121.1
1	33	4.8300	1.0716	23.1	2.38	33.0	0.53	2.9	1.2	-0.94	0.9	-489.1	644.3
1	43	3.3339	1.0716	25.9	2.38	22.7	0.53	4.0	2.3	0.74	0.8	383.5	1220.1
3	13	4.2275	1.0724	12.6	2.38	15.1	0.53	1.7	0.0	0.0	0.5	0.0	******
3	23	3.3011	1.0724	37.2	2.38	24.8	0.53	5.8	4.1	2.56	0.9	1202.6	1916.8
3	33	2.8782	1.0724	29.6	2.38	34.7	0.53	5.0	3.3	0.11	1.3	51.3	1559.2
3	43	3.9355	1.0724	30.6	2.38	36.0	0.53	4.3	2.6	0.08	1.2	36.7	1213.5
10	12	3.5632	1.0748	6.2	2.38	15.1	0.53	0.9	0.0	0.0	0.5	0.0	-0.0
10	22	4.4223	1.0748	9.3	2.38	15.2	0.53	1.2	0.3	0.39	0.4	0.0	-0.0
10	32	4.9613	1.0748	7.1	2.38	14.4	0.53	0.8	-0.0	0.14	0.4	0.0	0.0
10	42	5.0262	1.0748	7.7	2.38	14.0	0.53	0.9	0.0	0.23	0.4	******	-0.0

10	52	4.3424	1.0748	16.3	2.38	21.5	0.53	2.1	1.2	0.96	0.6	459.1	574.5
10	62	3.2867	1.0748	11.5	2.38	20.9	0.53	1.7	0.8	0.44	0.7	211.2	405.1
10	72	3.7347	1.0748	10.8	2.38	19.8	0.53	1.5	0.6	0.37	0.6	178.8	296.3
7	11	3.5745	1.0734	5.6	2.38	14.1	0.53	0.8	0.0	0.0	0.5	0.0	-0.0
7	21	3.0987	1.0734	17.8	2.38	22.9	0.53	2.8	2.0	1.39	0.8	******	******
7	31	3.3354	1.0734	9.5	2.38	18.4	0.53	1.4	0.6	0.33	0.6	******	******
7	41	4.2350	1.0734	8.3	2.38	17.5	0.53	1.1	0.3	0.18	0.5	0.0	-0.0
7	51	4.1885	1.0734	15.9	2.38	22.3	0.53	2.1	1.3	0.94	0.7	448.9	622.0
7	61	3.3511	1.0734	11.2	2.38	23.3	0.53	1.7	0.9	0.30	0.8	141.4	422.6
7	71	4.0071	1.0734	11.0	2.38	20.9	0.53	1.5	0.7	0.37	0.7	176.4	327.5
10	13	3.1890	1.0748	4.1	2.38	12.0	0.53	0.6	0.0	0.0	0.4	0.0	0.0
10	23	4.1113	1.0748	17.0	2.38	21.0	0.53	2.3	1.6	1.30	0.6	622.5	774.8
10	33	4.1839	1.0748	12.0	2.38	24.5	0.53	1.6	0.9	0.48	0.7	227.7	450.5
10	43	2.8120	1.0748	6.9	2.37	19.9	0.53	1.2	0.5	0.02	0.8	10.4	251.6
9	11	3.0734	1.0746	5.4	2.38	13.1	0.53	0.9	0.0	0.0	0.5	******	-0.0
9	21	3.6368	1.0746	12.0	2.38	19.0	0.53	1.7	0.9	0.60	0.6	******	******
9	31	3.1172	1.0746	5.8	2.37	12.3	0.53	0.9	0.1	0.12	0.4	******	******
9	41	3.4485	1.0746	4.3	2.37	12.0	0.53	0.6	-0.2	-0.09	0.4	3.0	******
9	51	3.6537	1.0746	17.8	2.37	19.2	0.53	2.6	1.7	1.44	0.6	625.6	745.4
9	61	3.6605	1.0746	13.2	2.37	22.5	0.53	1.9	1.0	0.57	0.7	249.8	454.4
9	71	3.4603	1.0746	9.8	2.37	19.8	0.53	1.5	0.6	0.25	0.7	109.0	261.4
10	11	4.3825	1.0748	3.9	2.37	11.7	0.53	0.5	0.0	0.0	0.3	******	******
10	21	3.7880	1.0748	15.2	2.37	24.6	0.53	2.1	1.6	0.98	0.8	******	0.0
10	31	3.0715	1.0748	8.1	2.37	16.3	0.53	1.3	0.8	0.42	0.6	3.2	0.0
10	41	3.2451	1.0748	5.5	2.37	14.1	0.53	0.8	0.3	0.12	0.5	48.5	0.0
10	51	4.6304	1.0748	15.5	2.37	20.3	0.53	1.9	1.4	1.09	0.6	520.4	684.1
10	61	3.5976	1.0748	9.6	2.37	21.2	0.53	1.4	0.9	0.36	0.7	174.2	420.8
10	71	3.3667	1.0748	8.5	2.37	20.0	0.53	1.3	0.8	0.27	0.7	131.3	369.1
9	12	3.0874	1.0746	4.7	2.37	10.8	0.53	0.8	0.0	0.0	0.4	******	0.0
9	22	2.6602	1.0746	4.8	2.37	10.0	0.53	0.9	0.1	0.08	0.4	-0.1	-0.0
9	32	3.6492	1.0746	5.7	2.37	12.1	0.53	0.8	0.1	0.06	0.4	0.0	-0.0
9	42	3.3202	1.0746	3.9	2.37	10.1	0.53	0.6	-0.2	-0.08	0.4	-0.0	******
9	52	3.2568	1.0746	15.8	2.37	17.5	0.53	2.5	1.7	1.27	0.6	553.7	740.1
9	62	2.5901	1.0746	11.0	2.37	20.0	0.53	2.0	1.2	0.41	0.8	180.2	534.6
9	72	4.0453	1.0746	12.4	2.37	19.2	0.53	1.7	0.9	0.55	0.6	240.4	408.2
8	12	4.0926	1.0725	5.0	2.37	13.2	0.53	0.7	0.0	0.0	0.4	******	-0.0

8	22	2.2205	1.0725	5.0	2.37	12.1	0.53	1.0	0.3	0.08	0.6	******	******
8	32	2.0217	1.0725	4.8	2.37	12.1	0.53	1.0	0.3	0.05	0.6	******	******
8	42	3.1721	1.0725	4.2	2.37	15.1	0.53	0.7	-0.0	-0.24	0.5	******	******
8	52	3.7368	1.0725	17.0	2.37	10.3	0.53	2.4	1.7	1.86	0.3	915.3	856.4
8	62	3.5104	1.0725	13.5	2.37	21.7	0.53	2.0	1.3	0.78	0.7	383.0	647.7
8	72	3.3642	1.0725	10.9	2.37	19.9	0.53	1.6	1.0	0.51	0.7	250.3	479.9
5	11	4.0004	1.0834	3.9	2.37	29.0	0.53	0.6	0.0	0.0	0.9	******	-0.1
5	21	3.6764	1.0834	24.1	2.37	12.5	0.53	3.6	3.0	3.35	0.4	******	******
5	31	3.5627	1.0834	11.9	2.37	21.0	0.53	1.8	1.3	1.38	0.7	******	-0.0
5	41	3.7092	1.0834	7.7	2.37	17.5	0.53	1.1	0.6	0.79	0.6	******	******
5	51	3.7697	1.0834	22.7	2.37	26.8	0.53	3.3	2.8	2.81	0.9	1287.4	1276.4
5	61	4.2135	1.0834	20.9	2.37	32.1	0.53	2.9	2.3	2.29	1.0	1048.5	1067.5
5	71	3.8421	1.0834	16.0	2.37	28.6	0.53	2.3	1.8	1.77	1.0	810.3	812.7
4	12	2.7869	1.0760	3.9	2.37	13.5	0.53	0.7	0.0	0.0	0.5	******	-0.0
4	22	3.6069	1.0760	6.4	2.37	15.0	0.53	0.9	0.3	0.30	0.5	******	-0.0
4	32	3.6981	1.0760	4.8	2.37	13.0	0.53	0.7	0.0	0.15	0.4	******	-0.1
4	42	3.5673	1.0760	4.3	2.37	12.4	0.53	0.6	-0.0	0.11	0.4	******	-0.0
4	52	4.1965	1.0760	28.0	2.37	28.0	0.53	3.7	3.1	2.67	0.9	1342.6	1550.3
4	62	3.5459	1.0760	19.8	2.37	30.3	0.53	2.9	2.3	1.63	1.0	821.7	1135.0
4	72	3.1535	1.0760	14.0	2.37	24.0	0.53	2.2	1.6	1.12	0.9	565.0	781.7
6	12	4.0592	1.0727	5.7	2.37	14.5	0.53	0.8	0.0	0.0	0.4	******	-0.0
6	22	3.1519	1.0727	12.0	2.37	18.1	0.53	1.9	1.1	0.77	0.7	******	******
6	32	2.9059	1.0727	9.0	2.37	0.0	0.53	1.5	0.7	1.49	0.0	******	0.0
6	42	3.4847	1.0727	9.9	2.37	0.0	0.53	1.5	0.7	1.47	0.0	******	-0.0
6	52	3.4584	1.0727	30.8	2.37	24.3	0.53	4.6	3.8	3.16	0.8	1633.8	1969.4
6	62	3.0160	1.0727	23.4	2.37	28.0	0.53	3.8	3.0	2.01	1.0	1037.9	1560.9
6	72	3.3273	1.0727	20.0	2.37	28.0	0.53	3.0	2.3	1.37	1.0	708.1	1175.8

TABLE I

STANDARD DEVIATIONS FOR INCREASED LACTATE AND EXCESS LACTATE

	ΔL			XL		
Treatment	Sum	Mean	S.D.	Sum	Mean	S.D.
11						
21	25.1	2.51	.92	18.14	1.81	1.63
31	10.6	1.06	.44	5.36	.54	.58
41	6.2	.62	.50	4.09	.41	.45
51	23.0	2.3	.88	17.3	1.7	.83
61	17.9	1.79	.91	9.8	.98	.86
71	13.5	1.35	.75	7.1	.71	.77
12						
22	7.1	.71	.51	4.4	.44	.68
32	2.4	.24	.39	2.1	.2	.61
42	.9	.09	1.03	1.5	.15	.59
52	25.0	2.5	.91	22.6	2.3	.94
62	19.8	1.98	1.1	12.5	1.25	1.15
72	14.1	1.41	.95	8.49	.85	.94
13						
23	34.2	3.42	1.51	25.4	2.54	1.95
33	24.0	2.4	1.28	11.6	1.16	1.32
43	20.0	2.0	1.04	11.5	1.15	1.1

APPENDIX C

MVO2 DATA

TABLE II

OXYGEN UPTAKE AT VARIOUS WORKLOADS FOR EACH SUBJECT

Workload	900	1050	1200	1350	1500	1650	1800
Subject							
01	2.37		3.35	3.31			
02	2.19		3.05	3.3	3.59	3.57	
03		2.6	3.11	3.45	3.59		
04	2.19		2.74	3.4	3.71	3.97	4.45
05	2.1		3.04	3.17	3.26	3.71	3.61
06	2.16	2.81	3.3	3.56	4.1		
07	1.98		2.73	2.97	3.43	3.91	
08		2.25		3.42	3.86 (1400)		
09	1.88	2.32	2.65	2.93	3.29	3.77	3.67
10		2.42	3.08	3.9	4.28		

Note: Workload in Kpms on Monarch bicycle ergometer.

Oxygen uptake in litres/minute.

APPENDIX D

OXYGEN EQUIVALENT DATA

TABLE III: COMPARING OXYGEN DEBT WITH OXYGEN DEBT EQUIVALENTS OF INCREASED LACTATE AND EXCESS LACTATE

		Treatment I			Treatment II			Treatment III		
S	O_2E	2	5	8	2	5	8	2	5	8
	O_2	2720			2494	1821	0	768	526	222
01	XL	599	221	58	945	545	321	606	-489	383
	L	824	558	417	1107	765	482	1121	644	1220
	O_2D					4050			2238	
	O_2	564	27	-54	-1979	-1946	-1919	1496	1113	566
02	XL	547	235	145	772	751	573	1424	793	503
	L	1098	803	543	977	977	764	1721	1300	961
	O_2D		2240			554			3006	
	O_2	620	1350	230	404	260	3	2760	2590	2002
03	XL	552	243	87	1700	1992	1258	1203	51	37
	L	1373	1240	867	1772	2217	1694	1917	1559	1214
	O_2D		1330			1834			4900	
	O_2	2292	1390	1118	-140	-460	-460	2208	902	272
04	XL	1237	867	625	1343	822	565	-808	13	-16
	L	1638	1359	1053	1550	1135	782	300	4	-82
	O_2D		4500			1910				
	O_2	950	470	368					879	479
05	XL	1287	1049	810	1652	-210	-390	1619	937	1571
	L	1276	1068	813	1230	420	3	1955	1384	1189
	O_2D		2880			774			1824	
	O_2	1256	616	396	-686	-696	-696	-164	-66	-126
06	XL	1760	1321	1189	1634	1038	708	2563	1688	1058
	L	2088	1857	1535	1969	1561	1176	2993	2320	1714
	O_2D		2526			774			1824	
	O_2	-802	-592	-642	3220	2710	2120	192	-108	-108
07	XL	449	141	176	899	550	388	2156	1049	910
	L	622	423	328	1079	910	687	2400	1637	1460
	O_2D		758			5500			2102	
	O_2	-448	-337	-232	880	640	556	-57	-117	-167
08	XL	575	242	152	915	383	250	1603	832	675
	L	743	509	354	856	648	480	1807	1296	1006
	O_2D		782			2380			1220	
	O_2	1950	1364	994	1033	467	228	1330	926	742
09	XL	626	250	109	554	740	240	1073	416	324
	L	745	454	261	180	535	408	1276	911	681
	O_2D		3620			2853			2790	

S	O_2E	Treatment I 2	5	8	Treatment II 2	5	8	Treatment III 2	5	8
	O_2	3460	2754	2363	1023	673	523	1460	1610	47
10	XL	520	174	131	459	211	179	623	228	10
	L	684	420	369	575	405	296	775	451	252
	O_2D		4990			2480			-	

Key to table.

S_____Subject

O_2E_____Oxygen equivalents

The 2, 5, and 8 columns_____time in minutes after exercise ceased and the figures in these columns represent the oxygen equivalents at these times.

O_2_____Oxygen debt figures obtained by direct collection of expired gas

XL_____Oxygen equivalent figures for excess lactate

L_____Oxygen equivalent figures for increase in lactate

O_2D_____Total oxygen debt for the treatment.

TABLE IV

OXYGEN DEBT MEANS FOR EACH TREATMENT

		Treatment I	Treatment II	Treatment III
Time	O_2E			
	O_2	1094	698	468
2min.	XL	831	1045	143
	L	1109	1129	1625
	O_2	671	382	820
5min.	XL	384	745	551
	L	869	957	1150
	O_2	505	-3.5	447
8min.	XL	348	409	545
	L	645	706	962

Note: figures represent total debt at that time period in millilitres.

APPENDIX E

BLOOD WATER DATA

TABLE V

BLOOD WATER FIGURES

Subject	Initial Wt test tube + blood	Final Wt test tube + blood	Wt of blood	Wt of blood solids	Blood Density	Blood Water
01	79.36	76.72	3.32	.69	1.59	2.64
02	80.45	77.67	3.48	.71	1.6	2.78
03	79.72	76.81	3.69	.78	1.58	2.91
04	79.61	76.99	3.28	.66	1.60	2.62
05	81.16	77.26	5.02	1.12	1.55	3.9
06	79.4	76.72	3.12	.62	1.61	2.5
07	79.39	76.61	3.48	.70	1.60	2.78
08	80.53	76.99	4.40	.866	1.60	3.53
09	80.42	76.71	4.63	.93	1.60	3.7
10	81.28	78.32	3.58	.67	1.62	2.9

Note: All weights are expressed in gms.

APPENDIX F

FORMS USED

UNIVERSITY OF ALBERTA
FITNESS RESEARCH UNIT and
FACULTY OF PHYSICAL EDUCATION

CONSENT TO PARTICIPATE IN A RESEARCH STUDY

SUBJECT. . . . . .DATE. . . . . TIME A.M. P.M.

1. I agree to participate in an investigation and in relation to this hereby authorize Drs . . . . . and or such assistants as may be selected by them, to perform the following procedure(s):

. . . . . .

. . . . . .

. . . . . .

2. Drs. . . . . have explained the purpose of this study and I understand the routine of the procedure outlined above.

. . . .
Witness Signature of Subject

If the subject is unable to sign or is under 21 years of age, complete the following:

The subject is a minor (. . . . . .years of age).

or

The subject is unable to sign because. . . . . .

As the closest relative or legal guardian I hereby sign on his/her behalf:

. . . . . .
Witness Signature Relationship

COMMENTS:

Name:

Weight:

Height:

Date:

Temp:

B.P.

STPD factor:

1. Oxygen, FeO_2 = (from Beckman)

   % = _____ / 40

   = _____ %

2. Carbon Dioxide, $FeCO_2$ = _____ %

3. Nitrogen = _____ %

4. VeSTPD = VeATPS + .30) x STPD factor

   = x

   = _____ litres/ min.

5. Volume of Inspired Air.

   = _____ x _____

   79.04

   = _____ litres/ min.

6. Oxygen Consumed. VO_2

   = (x .2039) - (x FeO_2)

   = _____ litres/ min.

. (Vol gas)

+ . _____ (STPD fac)

_____ X

. (Nitrogen)

+ . _____ X (VeSTPD)

.

-I.8978

_____ **

. **

+ I.3207

=

_____

. (FeO_2)

+ . _____ X

=

_____